Exhibit 2

Expert Opinion of Gail A. Van Norman MD

February 10, 2022

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I. Expert Qualifications (see also Addendum I: Gail A. Van Norman MD Curriculum Vitae):

I am a medical doctor, board-certified in both Internal Medicine and Anesthesiology, and subspecialty trained in Cardiovascular and Thoracic Anesthesiology The full details of my educational and professional background and experience, including my publications and invitational lectures can be found in the attached curriculum vitae as **Appendix I**.

A majority of my clinical experience has been based in cardiac and general anesthesiology, as well as perioperative care of the anesthesia patient; I have extensive personal clinical experience with high-dose benzodiazepine-based anesthesia for cardiac surgery patients in approximately 750 to 1000 cardiac cases.

Over the course of my career I have been an expert consultant/witness in 14 medically-related cases, both as a defense and as a plaintiff's expert in approximately equal numbers, and both as a paid and as a *pro bono* expert. In 3 of these cases, I was consulted as an expert in bioethics: i.e. regarding informed consent and end-of-life issues. In the remainder, I was consulted as an expert in the anesthesia care of a patient. In the last 4 years, I have provided testimony at trial or by deposition in 9 cases, 3 of which were lethal injection cases: **the list is included in Appendix II following this report.** I am not now, nor ever have been an employee of any legal firm or entity, nor do I derive a significant portion of my income from medico-legal expert witness activities. My fee schedule for this case is:

Review of medical records or other materials provided by Client	$400/hr
Phone conference, reference research, report preparation	$400/hr
Testimony, including deposition	$500/hr

II. Materials Reviewed

I have relied on my own significant clinical experience with the drugs in Oklahoma's three-drug protocol, including high-dose intravenous (IV) midazolam and other benzodiazepines (drugs of the

same class) used during the induction of anesthesia during cardiac surgery and other cases, as well as various authoritative textbooks, peer-reviewed articles, materials from government regulatory agencies such as the FDA, and other publications and materials as included in the references cited and footnoted throughout this report. I reserve the right to revise my opinion should further relevant information come to my attention, including productions relating to the executions conducted by Oklahoma in 2021 and 2022.

I have reviewed and relied upon the materials listed in **Appendix III: "Materials Relied Upon and/or Reviewed"** in preparing this report as well as the references cited in this report. More documents, studies, reviews and other pertinent information may become available to me prior to trial, and I reserve the right to take such materials into account and to modify/supplement my opinions accordingly. I may also be present at hearings or at trial, or review trial transcripts, and may take into account any testimony or other evidence presented there that is related to my opinions and modify/supplement my opinions accordingly.

III. The Oklahoma Execution Protocol

Oklahoma uses a 3-drug protocol for lethal injection executions consisting of 500 mg of midazolam, 100 mg of vecuronium bromide and 240 mEq of potassium chloride. Five minutes after administration of the first drug (midazolam) the protocol calls for a consciousness check by an IV team member. The specific steps of this consciousness check are not specified in the protocol, which only refers to "all necessary and medically appropriate measures". The declaration of the IV Team Leader, who I understand attended a U.S. accredited medical school, explains the methodology to be used simply as "verbal, sternum rub, painful stimulation." There is no definition of what would constitute confirmation of "unconsciousness" in the protocol. After this check, the remaining chemicals are injected in order: vecuronium, followed at an unspecified time by potassium chloride. Death is declared as the point where all electrical activity of the heart has ceased as indicated on a standard ECG monitor.

IV. Expert Opinion Questions

I have been asked to review the Oklahoma lethal injection protocol and the evidence of how it has been implemented to provide an opinion based on my medical expertise and understanding of the protocol of what a prisoner will experience as a result of this protocol. I intend to apply this analysis to the executions that have taken place for which we have documentation at the time I testify. This includes documentation on the execution of John Grant. I expect to receive documentation on the executions of Bigler Stouffer, Donald Grant and Gilbert Postelle prior to trial to include in my analysis.

My evaluation addresses the following questions:

First, what is midazolam's effect on consciousness and awareness and will a 500 mg dose render a prisoner insensate to the noxious stimuli of the protocol?

Second, what is the effectiveness and reliability of the protocol's consciousness check to establish a prisoner unconscious an insensate to such noxious stimuli?

Third, what are the effects of the vecuronium bromide and potassium chloride as used in the protocol and what noxious stimuli do they represent?

Fourth, what do the movements seen in prisoners during midazolam executions, including John Grant's execution, signify?

V. The Execution of John Grant

Sworn testimony of three eyewitnesses, Judy Gardner, Meghan LaFrancois, and Ervin Yen, a document entitled "J. Grant Correctional Service Log OAG - 019037-OAG – 019042", a hand-written document entitled "Grant OAG - 019353-OAG – 019353" that appears to have been contemporaneously created by an unnamed member of the Board of Corrections with times and comments during the execution, two media eyewitness reports and EKG/pulse oximetry tracings from the execution generally corroborate each other in the following sequence of events:

Prior to administration of midazolam[1]: At approximately 16:07-16:08 the inmate was awake and verbal.

16:09 (time 0): DOC service log indicates beginning of injection of chemicals. All witnesses agree that around 16:09 drugs started flowing, and the corrections official's note indicates that syringes 1A, 1B, and 1C were administered beginning at 16:09. These syringes were prescripted to contain a total of 500 mg of midazolam and a saline flush. Both Ms. Gardner and Ms. LeFrancois[2] testify that Mr. Grant continued to move purposefully at this point. He closed his eyes, lifted his head, opened his eyes and looked down toward his feet, then put his head back down and closed his eyes. He began to breathe heavily. Dr. Yen states in his testimony that he judged from 15 feet away and with no consciousness check that Mr. Grant was unconscious within 30-45 seconds.[3]

Mr. Grant's subsequent movements further disprove this. Dr. Yen states at one point that he judged Mr. Grant to be unconscious solely on the basis that he closed his eyes and wasn't "really moving a lot,"[4] neither of which demonstrates unconsciousness. He states that he agrees with the statement that "you can't be conscious and be unable to move muscles". Studies have shown that awareness in the operating room is *rarely* associated with movement. In one study that included both paralyzed and unparalyzed patients, less than 2% of patients who were aware actually moved, even if they were not paralyzed.[5]

1 minute after administration: Approximately 1 minute after the beginning of the drug administration, as documented in the correction official's notes and as testified to by both Ms.

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[1] Note that times recorded automatically on the EKG monitor are 13 minutes ahead of times recorded by the witnesses and at the time of death. I have taken this into account in my timeline, and have used the recorded time of death of 16:21 as an indication that the EKG machine "clock" ran 13 minutes ahead of actual time.
[2] Deposition of Meghan Francois, p 6
[3] Deposition of Dr. Ervin Yen, p 229
[4] Deposition of Dr. Ervin Yen, p 230
[5] Domino KB, Posner KL, Caplan RA. Awareness during anesthesia. A closed claims analysis. Anesthesiology 1999; 90:1053-61

Gardner, and Dr. Yen, the patient's airway began to partially obstruct. The signs of airway obstruction are clear. The handwritten note indicates that the inmate was "making noises". Ms. Gardner described that as Mr. Grant took deeper breaths, and his chest was still rising (so we know the airway was not yet fully obstructed) but he was having to make increasingly strong efforts to breath. Due to negative pressure in his chest resulting from his attempts to breathe against partial airway obstruction, he was also in the early stages of flash pulmonary edema. The EKG/pulse oximeter strips support this, demonstrating a decreasing oxygen saturation in Mr. Grant's blood at this time despite air movement.

It should be noted also that there is motion artifact present on the pulse waveform, along with failure of the pulse oximeter to correlate with the EKG, indicating he was moving. The actual readings recorded from the pulse oximeter (81 at 1 minute after injection of midazolam and of 73 at 2 minutes) are unreliable, because 1) there is obvious motion artifact on the tracing and it fails to follow the EKG, and 2) even more importantly, pulse oximetry readings in the low 80's and below do not correlate with measured oxygen levels in the blood.[6] However, both values would still be compatible with consciousness.[7]

Ms. Gardner stated that Mr. Grant began "gulping" air and his efforts to breathe became more pronounced and frantic. Ms. LaFrancois corroborates this, stating that at this point his breathing became "labored" and he began to cough, and he lifted his head off the gurney. Dan Synder reported that "his entire back lifted repeatedly off the gurney.[8] These types of movements are typical of patients with airway obstruction, including obstruction due to asthma, for example, and are part of efforts to reopen the airway. However, at this point, midazolam had caused the muscles in the upper airway (the tongue, the larynx, the glottis) to collapse, and had more than tripled the "work of breathing"—i.e. the amount of physical effort it takes to move air[9]; the airway obstruction progressed despite his efforts to breathe.

1-8 minutes: A few seconds thereafter, Ms. Gardner, Ms. Francois and Dr. Yen all agree that the Mr. Grant began making active movements, at times during the execution lifting parts of his body off of the gurney.[10,11,12,13] These movements were restrained by straps around his chest and arms. Ms.

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[6] Chiappini F, Pistelli LF. Accuracy of pulse oximeter in the measurement of the oxyhaemoglobin saturation. Eur Resp J 1998; 11:716-9

[7] Shaw D, et al. Hypoxic hypoxia and brain function in military aviation: basic physiology and applied perspectives. Front Physiol 2021; 17:12:665821

[8] Synder D. A minute-by-minute account of John Grant's death as told by witness of executions. Fox 25. KOH, Oklahoma City. Available at: https://www.msn.com/en-us/news/crime/a-minute-by-minute-account-of-john-grants-death-as-told-by-witness-of-execution/ar-AAQ4FbF Accessed Feb 10, 2022

[9] Montravers P, et al. Effects of intravenous midazolam on the work of breathing. Anesth Analg 1994; 79:558-62

[10] Deposition of Dr. Ervin Yen, p 232

[11] Deposition of Julie Garner, pp16,17

[12] Eyewitnesses report John Grant experienced repeated 'full body convulsions' and vomited during execution; Oklahoma says execution was carried out 'without complication'. Death penalty information center. October 29, 2021. Available at: https://deathpenaltyinfo.org/news/eyewitnesses-report-john-grant-experienced-repeated-full-body-convulsions-and-vomited-during-execution-oklahoma-says-execution-was-carried-out-without-complication Accessed Feb 20, 2022

[13] Snyder D. A minute-by-minute account of John Grant's death as told by witness of execution. Thursday Oct 28th, 2021. Available at: https://deathpenaltyinfo.org/news/eyewitnesses-report-john-grant-experienced-repeated-full-body-convulsions-and-vomited-during-execution-oklahoma-says-execution-was-carried-out-without-complication Accessed Feb 10, 2022

Gardner states that Mr. Grant began to vomit and "gasp for air" and states "I'd never seen anybody gasp for air like that in my life."[14] She states further in her affidavit that "he turned his head to the right and started vomiting."[15] According to the DOC service log, Mr. Grant began to vomit. All of the witnesses corroborate that vomit covered his face, then began to run down his neck and the side of his face, and also to the floor. After guards wiped the vomit off of his face, he began to convulse and vomited again.[16] Dan Snyder, anchor for Oklahoma City Fox News stated: "Almost immediately after the drug was administered, Grant began convulsing, so much so that his entire upper back repeatedly lifted off the gurney. As the convulsions continued, Grant then began to vomit." Multiple times over the course of the next few minutes medical staff entered the death chamber to wipe away and remove vomit from the still-breathing Grant."[17] Meghan LeFrancois states in her affidavit "I remember one specific point when Mr. Grant's back lifted dramatically up off of the gurney."[18]

Mr. Grant was strapped flat on his back to the gurney and, unable to clear the vomitus out of his airway, which was by this time running down the right side of his face and shoulder. During his gasps for air witnesses corroborate that the vomitus was visibly being sucked back in to his airway with his breaths, where it contributed to airway blockage below the glottis (deep in the back of the throat and trachea), and at this point he began to drown in his own vomit. Later findings by the medical examiner would show that Mr. Grant had actually sucked the vomitus all the way into the lower reaches of the airways (bronchi) during his strenuous efforts to breathe.

Dr. Yen states that he felt that this was passive regurgitation, which is belied by all of the other eyewitness descriptions as well as by standard medical terminology. Regurgitation is passive leakage of stomach contents into the esophagus as the lower esophageal sphincter relaxes. Vomiting, on the other hand, is the forceful retrograde expulsion of gastric contents from the body. It involves active propulsion of stomach contents and muscular contraction of the stomach[19], all of which were witnessed by multiple eyewitnesses at the time.

Mr. Grant raised his head off of the gurney during this episode of vomiting, and was retching and coughing so severely that several eyewitnesses termed his "violent" movements "convulsions". Ms. Gardner states that he also appeared to be choking.

More than 4 minutes after vomiting began, and 6 to 8 minutes after the beginning of the execution, the vomiting continued, and Ms. Gardner noted "labored breathing". At this point, the airway collapse is complete, the sensations of suffocation and drowning were accelerating, and efforts to breathe became

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[14] Deposition of Julie Gardner, p 10

[15] Declaration of Julie Gardner dated 11/17/21

[16] Eyewitnesses report John Grant experienced repeated 'full body convulsions' and vomited during execution; Oklahoma says execution was carried out 'without complication'. Death penalty information center. October 29, 2021. Available at: https://deathpenaltyinfo.org/news/eyewitnesses-report-john-grant-experienced-repeated-full-body-convulsions-and-vomited-during-execution-oklahoma-says-execution-was-carried-out-without-complication Accessed Feb 10, 2022

[17] Snyder D. A minute-by-minute account of John Grant's death as told by witness of execution. Thursday Oct 28th, 2021. Available at: https://deathpenaltyinfo.org/news/eyewitnesses-report-john-grant-experienced-repeated-full-body-convulsions-and-vomited-during-execution-oklahoma-says-execution-was-carried-out-without-complication Accessed Feb 10, 2022.

[18] Affadavit of Meghan Francois dated Nov 17, 2021

[19] Schiff, JH et al. Complications of managing the airway: vomiting, regurgitation and aspiration. IN; Hagberg and Benumof's Airway Management. 4th Ed. Elsevier Inc, Philadelphia PA. 2018. P868-89

more and more futile. The EKG tracings show continued heartbeat, and an oxygen saturation value of 43 appears on one strip (still compatible with consciousness). There is no pulse oximetry tracing, raising questions about motion artifact. However, aspiration of stomach contents can cause rapid oxygen desaturation.

Two members of the execution team entered the room and attempted to clean the vomitus out of Mr. Grant's airway and then left the room. He vomited again. A male member of the execution team entered the chamber again to clean his face. Ms. LaFrancois, Ms. Gardner and Dr. Yen all agree that Mr. Grant *turned his head from its position facing right, to a position facing left, and his shoulder moved as he did so*, showing that he was still sensate and still trying to clear his airway, but his efforts were getting weaker. Ms. Gardner states: "As the man entered, Mr. Grant raised his head and turned it towards us (to his left). He also tried to raise'move his shoulder but it was strapped down."[20] The man turned Mr. Grant's head to face up and midline, an action that would result in more vomitus entering his airway. This moment is corroborated by Dan Synder of Oklahoma City Fox News, who stated: "Multiple times over the course of the next few minutes medical staff entered the death chamber to wipe away and remove vomit from the still-breathing Grant."[21] No maneuvers resembling a consciousness check were observed by Ms. Gardner, Ms. LaFrancois, Dr. Yen, or media eyewitnesses.

16:15: At this point, according to the DOC service log, a member of the execution team declared that Mr. Grant was unconscious, although Ms. Gardner and Mr. Snyder state that Mr. Grant was still trying to breathe, a purposeful movement (turning his head away from the side of vomit) had occurred seconds before, and no consciousness check was witnessed by anyone who has given an account of the execution, including witnesses of the defendants, with the exception of Mr. Farris who has no medical training and was the DOC official present in the execution chamber.[22] He claimed a brief consciousness check was done that included looking at the eyes and doing a "sternal rub" which he does not describe. In contrast with all other events attested to during the execution, no other person testifying about the events corroborates his statement. In fact, at this point, Mr. Grant's movement to clear his airway should have been taken as a sign of consciousness.[23]

According to the DOC service log, injection of syringes 4A, 5A, 6A, 7A, 8A, and 9A proceeded from 16:15 to 16:19. The prescripted contents of these syringes was vecuronium, saline flush and potassium chloride, in order.

Also at 16:15, according to the DOC service log, vecuronium administration started, and Ms. Gardner noted that at this point, Mr. Grant exhaled deeply, his mouth opened, and his chest "settled", and his efforts to breathe abruptly stopped. In under 60 seconds the maximum clinical effect of vecuronium was reached. Mr. Grant was no longer able to move or breathe by 16:16, as evidenced by a lack of respiratory movement and pulse oximeter tracing, although he was almost certainly still sensate.

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[20] Affadavit of Julie Gardner, Nov 17, 2021

[21] Snyder D. A minute-by-minute account of John Grant's death as told by witness of execution. Thursday Oct 28th, 2021. Available at: https://deathpenaltyinfo.org/news/eyewitnesses-report-john-grant-experienced-repeated-full-body-convulsions-and-vomited-during-execution-oklahoma-says-execution-was-carried-out-without-complication Accessed Feb 10, 2022

[22] Richard E. Glossip et al. v. Randy Chandler et al. Transcript for motion of preliminary injunction before the Honorable Stephen P. Friot United States District Judge January. 10, 2022 p 189.

Dr. Yen has testified that he believed Mr. Grant was dead "well before 11 minutes,"[24] (this would also be before vecuronium and potassium administration was complete), which is an astounding statement to make, particularly since Dr. Yen was 15 feet away and did not conduct any examination of the prisoner for signs of life. And he is wrong. The DOC service log indicates that Mr. Grant was alive for another 5 minutes, and the EKG strips recorded during Mr. Grant's execution document that his heart stopped and death occurred merely a few seconds before the time of death was declared at 16:21, calling into question Dr. Yen's interpretation of what he witnessed.

VI. Expert Opinions and Their Bases

Summary Opinion: In my expert opinion the Oklahoma protocol as demonstrated by the recent executions in 2021 and 2022 is virtually certain to cause the prisoner to be aware of severe pain and noxious stimuli, such as sensations of drowning, suffocation, terror, and searing pain with injection of potassium chloride, while simultaneously paralyzing him or her so that there are few or no outward signs of pain and suffering.

The above-referenced summary opinion is supported by the underlying opinions and the bases therefore set forth below:

A. Movements of the prisoners during their executions, such as the movements of John Grant during a recent Oklahoma execution, are not "reflexes" but rather demonstrate pain and suffering throughout the execution.

Before the administration of muscle paralytic agents, and sometimes even during the onset of muscle paralysis, the extreme pain experienced during lethal injection executions has still stimulated some of the prisoners to make visible movements, which have been reported by the media and by other eyewitnesses. The vast majority of patients who are nevertheless aware, and even have recall under general anesthesia *will not move*, many of them even if they are not paralyzed and able to do so.[25] Eyewitnesses to midazolam executions have described convulsions, vocalizations, paradoxical motion of the abdomen, jerking of the body up and off of the gurney, coughing, choking, active vomiting and volitional turning of the head to clear the airway. These movements are not consistent with spinal reflexes, and clearly support the findings of the studies cited throughout this report. They demonstrate the prisoners' response to painful and excruciating feelings due to respiratory distress, sensations of drowning and searing pain during potassium chloride injection.

In John Grant's case, his movements demonstrated his struggle for air and sensations of drowning as he experienced the onset of airway obstruction, pulmonary edema, and the aspiration of stomach contents deep into his lungs.

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[24] Deposition of Ervin Yen, p 238

[25] Domino KB, Posner KL, Caplan RA. Awareness during anesthesia. A closed claims analysis. Anesthesiology 1999; 90:1053-61

Many other executions using midazolam also show movements indicative that the inmate was sensate during respiratory distress. The above-referenced movements of John Grant are examples of such movements indicative of being sensate, but there are numerous other examples in executions involving 500 mg administration of midazolam.

During the execution of Willie Smith in Alabama, which uses a 3-drug protocol consisting of midazolam, rocuronium, and potassium chloride, Spencer Hahn testified that he saw movements such as his arm jerking upward with injection of midazolam, his torso "bucking" off of the gurney and attempts to breathe that continued right up until the paralytic agent was given.

Hahn also witnessed the Alabama execution of Ron Smith, also using a 3-drug protocol consisting of midazolam, rocuronium and potassium chloride. He saw similar movements, and gasping "like a fish". After 500 mg of midazolam, Ron Smith failed to pass the consciousness check which consisted only of mild stimuli: calling the name, touching the eyelid and pinching the skin. *Despite a second 500 mg dose of midazolam Ron Smith again failed a consciousness check and responded to a light stimulus (skin pinch), but the execution proceeded nevertheless.*[26]

During the recent execution of Billy Ray Irick, using the same 3-drug protocol including midazolam employed by the state of Oklahoma, a witness described that, around "two minutes later", after the consciousness check, "Irick did appear to react physically to the second drug (vecuronium). He jolted and produced what sounded like a cough or choking noise. He moved his head slightly and appeared to briefly strain his forearms against the restraints."[27]

During the execution of Donnie Johnson, also using the same 3-drug protocol including midazolam that is used in the state of Oklahoma, he opened his mouth wide, made gurgling sounds for about 3 minutes, and then a higher-pitched gasp, according to one eyewitness.[28]

Jacob Rosenberg of The Guardian, for example, described the movements he witnessed of Marcel Williams in his execution in Arkansas (which employed the same 3-drug protocol involving midazolam used in the state of Oklahoma). He stated that during the execution, which began at 10:16pm: "[H]is back arched off the gurney as he sucked in air. I could not count the number of times his body moved in such a way, rising off the gurney. Protocol dictates that five minutes after the introduction of midazolam there should be no movements. But, at 10:21pm, Williams was still breathing heavily and moving."[29]

In the execution of Kenneth Williams in Arkansas, using the same 3-drug protocol including midazolam used in the state of Oklahoma, Kelly Kissel from the Associated Press stated that

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[26] Deposition of Spencer Hahn, page 45.

[27] Hale S. The execution of Billy Ray Irick. Nashville Scene. Aug 10, 2018. Available at: https://www.nashvillescene.com/news/pithinthewind/the-execution-of-billy-ray-irick/article_ef6c718d-bc1c-550f-926c-e68eb7fd9891.html Accessed Feb 10, 2022

[28] Tamburin A, Burgess K. 'No more dying there': death row inmate Don Johnson sang hymns as lethal drugs took effect. Commercial Appeal. May 17, 2019. Available at: https://www.commercialappeal.com/story/news/2019/05/17/donnie-edward-johnson-Oklahoma-execution-lethal-injection/3685417002/ Accessed Feb 10, 2022

[29] Rosenberg J. Arkansas executions: 'I was watching him breathe heavily and arch his back.' The Guardian, April 25, 2017. Available at: https://www.theguardian.com/us-news/2017/apr/25/arkansas-execution-eyewitness-marcel-williams Accessed Feb 20, 2022.

Williams "lurched 15 times in quick succession, followed by five slower lurches, three minutes after the sedative midazolam was introduced." Two other witnesses, Donna Terrell of Fox 16[30] and Knowles Adkisson of the Pine Bluff Commercial, corroborated the account. Donna Terrell stated that there "was a lot of heavy chest pumping and sounds coming out and the microphone was turned off and we could still hear. it."[31] She further stated that *even while Kenneth Williams continued to make sounds*, the attendant carried out a "consciousness check" and that the heavy breathing continued.

Comparison of the movements observed during the executions of these men to the movements that occur in dying patients, "spinal" reflexes, "posturing" , and "agonal respirations", clearly demonstrates that the movements were not "reflexes" and represent continued efforts to breathe despite airway obstruction right up to the point of chemical paralysis.

Involuntary "Spinal" Reflexes.

The term "involuntary reflexes" is actually not specific and can refer to several different types of movement in response to a given stimulus. Tapping your knee with a doctor's reflex hammer results in a little "kick" of the leg, for example. The "stimulus" for the reflex is a tap on a small tendon that runs between the kneecap (patella) and the tibia. The tap sharply stretches the tendon briefly, and nerve receptors detect the stretch and send a signal to the spinal cord, when then reacts locally in the low spine to send a signal back to kick the leg. The reflex does not involve any arcs in the brain, and therefore does not require nor indicate consciousness. Its primary purpose is to provide quick responses in the legs to changes in posture, balance, and leg coordination. Such movements require an inciting, usually specific stimulus, and are quick, isolated movements generally in only 1 or 2 muscle groups at a time (the knee extensor muscles in our example case). They are not generalized movements throughout the body. **Spinal reflexes occur in both the presence and absence of consciousness.** These generally quick, isolated muscle movements are very different than movements observed in the recent executions, which included "heaving", "convulsing", and raising up off of the gurney, all involving large muscle groups, and those particularly involved in respiratory distress, or in expressions of aversion.

Head Turning in Brain Death

Dr. Antognini has attempted to equate movements seen in the executions to "reflexes" seen in brain dead patients. Brain death is irrelevant to the topic of lethal injection executions: prisoners are not brain dead. In making this comparison, Dr. Antognini appears to think that if you are unconscious your nervous system works the same way would have the same reflexes that are present in a brain dead body. *But unconscious brains behave very differently than dead brains*

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[30] DeMillo A, Kissel K. Inmate convulses during deadline-beating execution, Arkansas' 4th in 8 days. NBC Miami. April 28, 2017. Available at: https://www.nbcmiami.com/news/national-international/arkansas-executes-4th-inmate-finishes-lethal-injection-schedule/1988323/ Accessed Feb 10, 2022

[31] Interview of Donna Terrell who witnessed Kenneth Williams' execution. April 27, 2017. Video available at: https://www.youtube.com/watch?v=BYf3eZZo_HE Accessed Feb 10, 2022

(indeed if that were not so, we would simply be able to declare that anyone who is unconscious is dead).

Brain dead bodies do sometimes demonstrate stereotypical, repetitive movements that are spinal cord mediated called "spontaneous reflex movements" or SRMs. These movements can include a stereotypic type of repetitive head turning. The Wu article[32] referenced by Dr. Antognini was a letter describing a case of head turning in a presumably brain-dead patient, which the authors admit may *not* be due specifically to spinal cord reflexes, and may involve brain-centered mechanisms (which by definition cannot occur in brain dead bodies). Researchers who have studied SRMS, including authors cited by Antognini, agree that SRMs likely occur precisely *because* the brain is dead, and because brain activity that would otherwise be present isn't suppressing signals in the spinal cord that cause these movements.[33,34,35]

Posturing

In patients who have suffered injury in the brain, more complex, stereotypic movements can occur in multiple muscle groups at once in response, generally, to painful stimuli. These movements are widespread and cause the body and/or limb position to change in a very predictable way. Typically, the patient appears to lie unconscious, but when pinched on the foot, for example, simultaneously stretches all limbs slightly, turning the wrists down toward the mattress, pointing the toes toward the mattress and arching the head and neck slightly back. In a slightly different type of posturing, the toes are pointed down and the arms held tightly bent at the elbows up on the chest.

Because these postures were first described in patients in coma, they were termed "decerebrate" or "decorticate" posturing, and they were believed to indicate a lack of activity in the cortical (i.e. "thinking") areas of the brain and therefore to indicate a loss of awareness or consciousness —a misconception that persists among many experienced anesthesiologists and other clinicians. Even early on, clinicians reported that **many patients with decerebrate/decorticate posturing were conscious.**[36,37,38,39,40,41] Largely due to the development of advanced neuroimaging (e.g. magnetic resonance imaging [MRI] and positron-emission tomography [PET scanning]), we now know that decerebrate and decorticate posturing is *not* indicative of unconsciousness, and have been clearly demonstrated in conscious people. Moreover, MRI scans demonstrate preserved consciousness in patients with decerebrate/decorticate posturing who are unable to interact with their

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[32] Wu Y, Balaguer PO. Spontaneous and reflex head turning in brain death. Crit Care 2013; 17:440.
[33] Saposnik G et al. Movements in brain death: a systematic review. Can J Neurol Sci 0209; 36:154-60
[34] Jain S and DeGeorgia M. Brain death-associated reflexes and automatisms. Neurocrit Care 2005; 3:122-6
[35] Wu Y, Balaquer PO. Spontaneous and reflext head turning in brain death. Crit Care 2013; 17:440
[36] Halsey JH, Downie AW. Decerebrate rigidity with preservation of consciousness. J Neurol Neurosurg Psychiatry. 1966; 29:350-5
[37] Feldman H. Physiological observations in a chronic case of "locked-in" syndrome. Neurology 1971; 21:459-78
[38] Pattisapu J, Smith RR, Bebin J. Traumatic decerebracy with preserved consciousness and voluntary movement. Neurosurgery 1985; 16:71-4
[39] Mahapatra AK, Bhatia R. Preservation of consciousness in a decerebrating head injured patient. J Assoc Physicians India 1990; 38:305-6
[40] Damasceno BP. Decerebrate rigidity with preserved cognition and gait: a possible role of anoxic-ischemic brain damage. Int j Neurosci 1991; 58:283-7
[41] Kao CD, Chen JT, Lai KL, et al. Gabapentin for decerebrate rigidity: a case report. Clin Drug Invest 2008; 28:67-70

environment.[42] Patients with decerebrate/decorticate posturing must be presumed conscious unless proven otherwise.[43]

Changes in facial expressions, blinking, breathing, moaning, opening and closing the mouth, moving the eyes or lips, lurching, convulsing, vomiting and other movements seen in prisoners during execution by lethal injection using midazolam are *not* elements of decerebrate or decorticate posturing.

Agonal Respirations/Cheyne-Stokes Breathing/Gasping

A number of changes in breathing occur in patients who have abnormal brain function, including drug-induced sleepiness, traumatic brain injury, and lack of oxygen to the brain. Cheyne-Stokes breathing is a pattern of respirations in which breathing first becomes faster and deeper, then gradually shallower and slower, until breathing stops altogether, but then resumes spontaneously after a period of no breathing. The pattern repeats over and over, usually follows a cycle of 30 seconds to 2 minutes. It is not an indicator of unconsciousness, and can occur in either conscious or unconscious patients. When this pattern occurs in conscious patients, the patients also report symptoms of shortness of breath, and inability to "get enough air" but despite this, they "don't just breathe" to relieve the sensations.[44]

Agonal respirations, a different type of breathing that can occur in dying patients, is also known as "terminal gasping" and appears to occur in all mammalian species. The respiratory center in the medulla of the brain begins to fire in response to decreased oxygen, forcing gasping movements involving contraction of the diaphragm. These contractions are not believed to be conscious or voluntary. The mouth is open during agonal respirations, and air movement into the mouth and trachea in response to these contractions may lead to short, brief sounds that resemble hiccups. Longer moaning, sighing, turning of the head, movements of the arms or other areas of the body are not consistent with agonal respirations.

The sequence of events in human asphyxiation (strangling or suffocation) has been established through study of filmed hangings (suicides, autoerotic accidents, homicides, and executions). Agonal respiratory movements are very short-lived, and prolonged agonal respirations do not occur.[45,46] Prolonged efforts to breathe during lethal injection executions do not represent agonal reflexes.

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[42] Kao CD, Guo WY, Chen JT, Wu ZA, Liao KK. MR findings of decerebrate rigidity with preservation of consciousness. Am J Neuroradiol 2006; 27:1074-5

[43] Latronico N, Antonini L, Taricco M, Vignolo LA, Candiani A. Approach to the patient in vegetative stat: part II: differential diagnosis. Minerva Anestesiol 2000; 66:233-40

[44] Leonard J. What's to know about Cheyne-Stokes respiration? Medical News Today. May 6, 2017. Available at: https://www.medicalnewstoday.com/articles/317313.php Accessed Feb 10, 2022

[45] Sauvageau A, LaHarpe R, Geberth VJ, et al. Agonal sequences in eight filmed hangings analysis of respiratory and movement responses to asphyxia by hanging. J Forensic Sci 2010; 55:1278-81

[46] Sauvageau A, LaHarpe R, King D, et al. Agonal sequences in 14 filmed hangings with comments on the role of the type of suspension, ischemic habituation, and ethanol intoxication on the timing of agonal responses. Am J Forensic Med Pathol 2011; 32:104-7

Respiratory efforts in a conscious patient with an obstructed airway, in contrast to agonal respirations, are characterized by forceful efforts to breathe that lead to minimal or no air passage (because the upper airway is obstructed). This results in something called "chest-abdominal paradox,"[47] a phenomenon well-recognized by anesthesiologists and sleep medicine specialists as a physical sign that the airway is blocked. This is *not* a terminal breathing pattern or "dying reflex".

In normal, unobstructed respiration while laying supine (on one's back) during inspiration, the diaphragm contracts downward into the abdomen. The abdomen rises because the diaphragm pushes the abdominal contents upward. At the same time, the movement of the diaphragm creates a vacuum in the chest, and under normal circumstances air rushes in through the trachea to fill the extra space created in the chest when the diaphragm contracts. Thus, the chest rises together with the abdomen as the chest expands with the inrushing air. In chest-abdominal paradox, however, the movements of the chest and abdomen become disconnected. The diaphragm contracts, moving into the abdomen and pushing the abdominal organs upward, causing the abdomen to rise. But the upper airway is obstructed and no air enters the chest. As the diaphragm pulls down into the abdomen, the vacuum it creates sucks the chest wall down with it. In this sequence the chest collapses inward while the abdomen is rising—giving the appearance of the chest and abdomen rocking opposite of one another, or a heaving abdomen. If the sequence continues, recruitment of chest wall muscles to breathe more forcefully and pull air into the chest causes the chest to rise, but again no air is entering the chest because the upper airway is obstructed. This motion causes the vacuum in the chest to increase, overcoming the pull of the diaphragm downward. The diaphragm is then "sucked" back toward the chest to fill the space where air should go, and the abdominal organs fall inward and are sucked upward toward the chest as well. The chest rises, but at the same time the abdomen collapses inward, and rocking in the opposite direction occurs. This gives rise to a "heaving" motion, which increases as the efforts to breathe become more and more violent. The sequence repeats, often with increasing recruitment of accessory muscles, and deepening of the heaving, until either the airway opens, or the muscles finally lose strength and then cease to move altogether.

***None* of the descriptions above of the eyewitnesses to lethal injection executions is compatible with dying "reflexes". The movements are, however, classic for active efforts to breathe against a closed upper airway and consistent with both chest-abdominal paradox, and voluntary movements in a sensate inmate feeling the onset of paralysis, suffocation or drowning in vomitus during the development of pulmonary edema.**

In some executions, inmates have demonstrated far less dramatic movements than those described above. However *whether there is movement or lack thereof*, human studies show that midazolam does not make the inmate insensate, and so-called consciousness checks will not be able to detect that consciousness is present.

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[47] Berry R. Monitoring respiration—event definition and examples. In: Fundamentals of Sleep Medicine. Saunders/Elsevier Inc. Philadelphia PA. 2012. pp 119-140

B. Midazolam is not an anesthetic and it has been shown repeatedly in human studies that it does not as a solo drug prevent awareness or responsiveness when severely painful stimuli are applied. It will not render an inmate insensate to severe pain.

Midazolam is a member of the benzodiazepine class of drugs, which are used primarily as sedative/hypnotic (sleep-producing) agents. **Midazolam has no analgesic (pain-relieving) properties.** This is not opinion, but scientific fact, as is confirmed by all authoritative texts, as well as an extensive literature.[48,49] To quote Goodman and Gillman, one of the most authoritative texts in clinical pharmacology and therapeutics in the world:

> "Although the clinical literature often refers to the 'anesthetic' effects and uses of certain benzodiazepines, these drugs do not cause a true general anesthesia; awareness usually persists, and a failure to respond to a noxious stimulus sufficient to allow surgery *cannot be achieved.*" [my italics]

As a solo drug, administration of midazolam does not and cannot render a person insensate or result in a state of general anesthesia, which is defined as a combination of analgesia or "antinociception" (lack of pain), amnesia (lack of recall), lack of awareness, and immobility with significantly painful or noxious surgical stimulation.[50] I know of no reputable anesthesiologist who would rely on it as a sole drug for general anesthesia for a significantly painful procedure. In fact, anesthesiology researchers have determined that it is useful only as a sedative for minor procedures. It is used, albeit rarely, as a solo *sedative*[51] in some low-discomfort procedures such as colonoscopy—which is of such low stimulus that it is performed in the majority of patients in the United States without any sedation at all.[52] In the vast majority of those cases that do involve the use of midazolam, it is combined with a powerful narcotic, such as fentanyl. The use of midazolam in such procedures is predicated note on the theory that midazolam will perform as a solo anesthetic, but rather to prevent the patient from remembering any pain they experience.

Midazolam is best known for its amnestic effect—i.e. its ability to repress memory, and this effect was a subject of significant interest even before its approval by 1988. Recent studies find that midazolam, when administered in the preoperative holding area increases amnesia for the preoperative holding area and/or the operating room[53].

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[48] Hemmings HC Jr, Egan TD. Pharmacology and physiology for anesthesia. Sanders Inc 2013. pp 144-6

[49] Mihic SJ, Mayfield J, Harris RA. Chapter 19: hypnotics and sedatives. IN: Brunton L, Hilal-Dandon R, Knollmann BC, Ed. Goodman and Gillman's the Pharmacologic Basis of Therapeutics 13th Ed. McGraw-Hill Education. 2018.

[50] Brown, EN, Purdon PL, Akeju O, Solt K. Monitoring the state of the brain and central nervous system during general anesthesia and sedation. IN: Miller's Anesthesia. Gropper MA, Miller RD, Eds. Elsevier Inc, Philadelphia PA. 2021. p1279

[51] "Sedative" differs from general anesthesia in that the patient is drowsy, and may even lightly sleep, but is arousable with stimulation, and will move and respond. Under general anesthesia, there patient does not respond to even severe stimulus, although they may still be aware.

[52] Hoffman MS, Butler TW, Shaver T. Colonoscopy without sedation. J Clin Gastroenterol 1998; 26:279-82

[53] Chen Y, Cai A, Dexter F, et al. Amnesia of the operating room in the B-Unaware and BAG-RECALL clinical trials. Anesth Analg 2016; 122:1158-68

The intended and unintended effects of midazolam are highly dependent on factors related to the pharmacokinetic characteristics of the drug (how quickly is it metabolized, and/or redistributed to places in the body, such as fat, where it will have no pharmacologic [clinical] effects); the method of administration of the drug; the individual to whom it is administered; *and severity of the surgical or other stimulus applied.*

When the FDA approved use of midazolam, it issued approval for its use as "*an*" anesthetic induction agent, not "*the*" anesthetic induction agent. It furthermore did not approve the drug as an "anesthetic" agent—for use to maintain general anesthesia. The FDA states that is allowable to administer midazolam for induction of anesthesia *before administration of other anesthetic agents*,[54] and merely states in packaging that it can be the first of several drugs administered during anesthetic induction.[55] In other words, it is permitted by the FDA for midazolam to be used in combination with narcotics, sedatives, and hypnotics (sleep-inducing drugs) such as propofol, barbiturates, or inhaled anesthetic agents to induce anesthesia. The FDA packaging explicitly states that midazolam can achieve anesthetic induction *"with the use of narcotic premedication"* (i.e. it needs to be combined with other anesthetic drugs) and can be used "*as a component of intravenous supplementation of nitrous oxide and oxygen (balanced anesthesia)*."[56] The FDA did not approve midazolam as a sole agent for induction, or more importantly, for maintenance of anesthesia during a severe stimulus.

While benzodiazepines may be listed as the "cause of death" in drug overdose cases, the actual "mode" of death is not due to direct drug toxicity. Midazolam has no direct deleterious effects on cells in the body, and produces no irreversible effects, but can temporarily interfere with the body's normal responses, such as breathing more deeply in response to hypoxemia (lack of oxygen) or hypercarbia (high levels of carbon dioxide in the blood). This is in contrast to drugs with intrinsic lethal toxicity, such as cyanide, which irreversibly stops oxidative function of cells. No "fatal dose" of midazolam in humans has ever been scientifically proven,[57] whereas high doses of anesthetics are usually lethal. Potential mechanisms of death in situations that involved midazolam administration are generally centered around respiratory issues after administration of midazolam in combination with other agents. Midazolam by itself causes only minimal and temporary slowing of breathing. In fact, as described by eyewitnesses to executions using massive

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[54] US FDA Center for Drug Evaluation and Research: Approval Package for Midazolam. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/nda/2012/075293orig1s014.pdf Accessed Feb 20, 2022

[55] Ibid.

[56] Ibid.

[57] Schultz M, Iwersen-BergmannS, Andresen H, Schmoldt A. Therapeutic and toxic blood concentrations of nearly 1,000 drugs and other xenobiotics. Crit Care 2012; 16:R136

doses of midazolam, prisoners generally do not stop struggling to breathe until the paralytic drug is administered.[58,59,60,61,62]

All benzodiazepines have similar a mode of action, and generally differ from each other primarily in water solubility, how much pain they cause during IV injection, rapidity of onset of action, duration of action, and the relative potency of a milligram of drug. Most commonly prescribed in small doses as an anxiolytic (i.e. to reduce anxiety), midazolam has **never** been widely used as the solitary hypnotic ("sleep-producing") component in general anesthesia (which commonly entails the administration of 6 to 10 different drugs). There are no controlled clinical studies that involved the administration of 500 mg of midazolam to a human subject. In order to comment on the effects of midazolam as a *solo drug* in preventing awareness, pain, and suffering during a severely painful or stimulating event, we therefore have to rely in part on studies that have been done in another very closely related and nearly equipotent[63] benzodiazepine, diazepam (also known by its trade name Valium), in addition to a few studies done using midazolam itself.

Diazepam was used briefly as a primary hypnotic for heart surgery in the 1980s and early 1990s, with poor results even though its use was with other drugs and not as a sole drug. Due to its variability of onset, unreliable duration of sedative action, lack of reliable production of complete unresponsiveness during surgery, undesirable effects on blood pressure (hypotension), and the severe pain it produces when injected intravenously, valium is no longer commonly used in modern anesthesia practice, except perhaps in isolated geographic areas.[64] Like midazolam, the pharmacokinetics (how the drug is taken up, metabolized and cleared from the body) of IV diazepam are unaffected by dose, even at very high doses.[65] As with midazolam, prior or concomitant prescription or illicit drug use by the recipient can lead to cross-tolerance (requiring markedly higher doses to produce its usual effect), or to synergistic actions. Paradoxical reactions to both of these benzodiazepines are common and include delirium, combativeness, paradoxical fear, anxiety and panic.

Action of Midazolam in the Brain

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[58] Deposition of Spencer Hahn

[59] Snyder D. A minute-by-minute account of John Grant's death as told by witness of execution. Thursday Oct 28th, 2021. Available at: https://deathpenaltyinfo.org/news/eyewitnesses-report-john-grant-experienced-repeated-full-body-convulsions-and-vomited-during-execution-oklahoma-says-execution-was-carried-out-without-complication Accessed Feb 10, 2022

[60] Deposition of Julie Garner

[61] Deposition of Meghan Francois

[62] Rosenberg J. Arkansas executions: 'I was watching him breathe heavily and arch his back.' The Guardian, April 25, 2017. Available at: https://www.theguardian.com/us-news/2017/apr/25/arkansas-execution-eyewitness-marcel-williams Accessed Feb 10, 2022.

[63] Equipotency indicates that a one drug produces virtually identical effects as a different drug, usually of the same class, at the same dose. Thus, 1 milligram of midazolam behaves in all critical ways almost identically to a 1 milligram dose of diazepam.

[64] Author's note: in fact, because the use of a benzodiazepine as the sole hypnotic agent during induction as described in the Oklahoma Protocol is so archaic to modern anesthesia practice, references regarding the efficacy and side effects of this combination necessarily rely on literature from the 1980s and 1990s. In addition, as a cardiac anesthesiology specialist, this Expert has had past personal extensive clinical experience in approximately 750 to 1000 cases with use of benzodiazepines as a sole hypnotic agent, a practice which was widespread in anesthesia for heart surgery in the 1980s.

[65] Ochs HR, Greenblatt DJ, Lauven PM et al. Kinetics of high-dose i.v. valium. Br J Anaesth 1982; 54:849-52

"Neurotransmitters" are the chemicals largely responsible for transmitting information in the brain and peripheral nervous system. They are manufactured and stored by nerve cells and released in response to various conditions in the cell's environment. These chemicals can be excitatory (causing increased metabolic, electrical and chemical activity in the cells they reach) or inhibitory (reducing the metabolic, electrical and chemical activity in the cells they reach). A balance of excitatory and inhibitory influences govern how the brain, spinal cord and nerves will react to a given stimulus. The mechanisms of actions of many different "sedative" drugs on the brain generally fall into 2 categories: 1) enhancement of the actions of inhibitory neurotransmitters and 2) blockade of the actions of excitatory neurotransmitters. The mechanism of action of midazolam in the brain is essentially confined to its effects on a single receptor, the "GABA" receptor.

GABA (gamma aminobutyric acid) is the primary *inhibitory* neurotransmitter in the mammalian central nervous system (i.e. brain and spinal cord), and acts at specific GABA receptor sites found in the brain. Drugs that enhance GABA effects or that have stimulatory action on the GABA receptor are referred to as "GABAergic" drugs. A single receptor for GABA, the $GABA_A$ receptor, is the only proven site of action of midazolam in producing sedation. The drug behaves like a key in a lock: once the receptor is bound, no more molecules of benzodiazepines can "join in". The receptor is "locked".

These receptors are present throughout the brain and affect function in the cerebral cortex, thalamus and hypothalamus, and other brain structures involved in consciousness. The critical implications of midazolam's actions at this receptor, on how the receptor creates a ceiling effect for midazolam, and on the resulting effects on consciousness, responsiveness, and recall, are discussed in more depth later in this report.

Midazolam Does Not Produce Unconsciousness During Severely Painful Stimulation

Many clinicians, even experienced and expert anesthesiologists, erroneously use the terms consciousness, awareness, and recall interchangeably, and/or rely on studies and data regarding *recall* when discussing *consciousness and/or awareness* under anesthesia.[66,67] However, these terms mean different things, involve entirely different brain functions, and cannot be used interchangeably. ***Lack of recall does not equal lack of consciousness, and lack of responsiveness does not indicate unconsciousness.***[68]

Consciousness, Responsiveness and Recall, and Midazolam

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[66] Absalom A, Nagels W. Fentanyl and midazolam anaesthesia for coronary artery bypass surgery. [letter to the editor]. Br J Anaesth 2000; 85:940-1

[67] Russel IF. Midazolam-alfentanil: an anaesthestic? An investigation using the isolated forearm technique. Br J Anaesth 1993; 70:42-6

[68] Sanders RD, Tononi G, Laureys S, Sleigh JW. Unresponsiveness ≠ unconsiousness. Anesthesiology 2012; 116:946-59

In order to understand issues concerning the 3-drug protocol and why it will not prevent a prisoner from experiencing extreme pain and suffering during lethal injection, it is important to have an accurate understanding of the different concepts of **consciousness, awareness, responsiveness** and **recall.**

The science of consciousness has advanced rapidly in the last five to ten years, with more and more functional testing of the conscious and unconscious brain now possible using technologies such as functional MRI scanning (fMRI). This has led to a fuller understanding of brain connectivity and consciousness, and what drugs that produce unresponsiveness do and do not do for consciousness.

The Isolated Forearm Technique (IFT)

Researchers into awareness under general anesthesia recognize that the "gold standard" for studying consciousness after injection of various "sleep" drugs during general anesthesia is the isolated forearm technique (IFT).[69,70,71,72,73] In the IFT, prior to administration of a paralytic drug, each patient has one forearm isolated from the circulation by a tourniquet to prevent the arm from being paralyzed. This allows the patient to move that arm in response to commands if they are awake and sensate. The ability to follow a specific command (such as "move your right arm", or "raise your right hand") *requires a high level of consciousness and awareness*: the patient must hear the instruction, realize what they are being told to do (attend to the event), and carry out the action in the instruction. Thus, a purposeful response on the IFT incontrovertibly demonstrates a high level of consciousness and awareness and indicates that the individual is sensate. Conversely, however, a lack of response *does not* demonstrate that the person is unconsciousness or insensate. It has been demonstrated that some patients who do not move on testing with the IFT nevertheless can recall explicit details of the surgery.[74,75,76] Thus, movement on the IFT proves consciousness and awareness, but lack of movement does not mean that the patient is necessarily unconscious.

The term "**consciousness**" describes a state of having a subjective experience of something. Consciousness can be "connected" or "disconnected." **Connected consciousness**—also called **awareness**—is the subjective experience of an event related to the "real world" environment in a

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[69]Linassi F, Zanatta P, Tellaroli P, Ori C, Carron M. Isolated forearm technique: a meta-analysis of connected consciousness during different general anaesthesia regimens. Br J Anaesth 2018: 121:198-209

[70] Zand F, Hadavi SM, Chohedri A, , Sabetian P. Survey on adequacy of depth of anaesthesia with bispectral index and isolated forearm technique in elective Caesarean section under general anaesthesia with sevoflurane. Br J Anaesth 2014; 112:871-8

[71] Jessop J, Jone JG. Conscious awareness during general anaesthesia—what are we attempting to monitor? Br J Anaesth 1991; 66:635-63

[72] Barker I, Deeprose C, Andrade J. Response to editorial by Davidson. Paediatric Anaesth 2003; 13:644

[73] Jones JG, Aggarwal SK. Monitoring depth of anesthesia. In: Gohneim MM, ed. *Awareness during anesthesia.* Oxford. Butterworth-Heinmann, 2001; 69-92

[74] Russell IF. Conscious awareness during general anaeshtsia: relevance of autonomic signs and isolated arm movements as guides to depth of anaesthesia. In: Jones JG, ed. *Bailliere's Clinical Anaeshtesiology, vol 3, Depth of Anaesthesia.* London: Bailliere-Tindall 1989, pp511-32

[75] Russell IF, Wang M. Absence of memory for intraoperative information during surgery under adequate general aneaaesthesia. Br J Anaesth 1997; 78:3-9

[76] Sanders RD, Tononi G, Laureys S, Sleigh JW. Unresponsiveness ≠ unconsiousness. Anesthesiology 2012; 116:946-59

[76]Linassi F, Zanatta P, Tellaroli P, Ori C, Carron M. Isolated forearm technique: a meta-analysis of connected

physical, emotional, or psychological way. Awareness is present, for example, when an awake person savors a meal. They are having a subjective experience that is directly related to a "real world" occurrence. **Disconnected consciousness** usually occurs when we are dreaming and are unaware of our "real world" environment.[77] Dreams are also subjective experiences—we might remember them when we wake, may have intense emotional reactions to them, and may even respond physically to them by moving and vocalizing in our sleep—but the dreamer is not usually reacting to what is happening to them in the physical world. When the physical world intrudes, and the dreamer "awakes," they move from disconnected consciousness into connected consciousness—aware of and responding to their environment once again.

Unfortunately, **consciousness** is often confused—even by experienced anesthesiologists—with **responsiveness**, although these terms refer to entirely different phenomena. While consciousness is the subjective experience of something, *responsiveness* is the ability to react physically to a conscious experience. Consciousness and responsiveness do not go hand in hand. A person can have connected consciousness and be sensate, and yet be unable to respond and demonstrate that consciousness[78] — and there are many examples in and outside of anesthesia. Unresponsiveness can occur in a conscious person due to 1) physical impediments, such as being paralyzed by a drug such as rocuronium, that immobilizes the muscles so that the surgeon can work within the belly or chest or pelvis; 2) nonphysical impediments, such as fear or surprise, e.g. being "frozen" by terror; and 3) physiological problems within the brain itself, e.g. strokes and drug effects that interrupt outgoing nerve signals and prevent purposeful movement even though muscles are not paralyzed and the person is conscious of their surroundings and can feel pain (a state referred to as "locked-in syndrome").

In considering the actions of a drug like midazolam during judicial lethal injection, we are concerned primarily with "connected consciousness"—the subjective experiences of the prisoner in his/her physical environment—such as whether they are able to experience pain or other sensations such as suffocation, despite being unresponsive. A lack of responsiveness in no way indicates a lack of connected consciousness, as a vast amount of human research now shows.

The scientific concept of consciousness in all of its forms is difficult to identify at best. Many definitions of consciousness emphasize the activity in the cortices of the brain (rather than the brainstem which controls more or less spontaneous functions, such as breathing and heartbeat, among others) in which a wide variety of information is integrated in a manner that is "irreducible," meaning the brain cannot ignore aspects of it. As one author describes it "when you open your eyes....you can't simply choose to see everything in black and white"—the brain integrates into your experience all of the colors (presuming that you have normal vision).[79] The brain weaves together multiple sensory experiences, together with the thoughts, emotions, and reactions that the experiences elicit in the cortices of the brain, and presents it to the person in one seamless event.

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[77] The special case of "lucid" dreaming is a state in which a person is dreaming, but probably does have connected consciousness. They are aware they are dreaming, and can communicate to the external world. It is not known if they will take commands and much more research is needed to fully define this state of consciousness as either connected or unconnected.

[78] Sanders RD, Tononi G, Laureys S, Sleigh JW. Unresponsiveness ≠ unconsciousness. Anesthesiology 2012; 116:946-59

[79] Koch C. What is consciousness? Scientific American. June 1, 2018. Available at: https://www.scientificamerican.com/article/what-is-consciousness/ Accessed July 9, 2018

The "cortex" of the brain (the so-called "gray matter" that makes up a large part of the brain and gives the brain its traditional shape) has traditionally been thought of as ruling thought, consciousness, and responsiveness. However, it turns out that much deeper core brain structures and their interactions with the cortex are equally critical to consciousness. Much of the current research regarding mechanisms of consciousness focus on the thalamus and other structures, such as the amygdala.

The thalamus lies buried deep within the core of the brain and is responsible for receiving and selecting among the billions of bits of data and sensory input received by the nervous system every moment. It processes data and sensory input by selecting some and rejecting others, and then connects with the cortex as well as arousal centers deeper in the brain to integrate consciousness of and responsiveness to those inputs. The thalamus thus behaves much like a giant, multimodal switching center in a hugely complex railyard of electrical signals and inputs, sending millions of signals at nearly the speed of light along some tracks to the cortex and others along different tracks to arousal centers or other areas of the midbrain for processing, and sometimes simply allowing some signals to be extinguished. Along the way, the signals that travel the tracks are amplified or dampened by additional signaling and chemicals in the brain. Interference with signal transmission in the thalamus does not appear to affect consciousness, but does affect responsiveness. Strokes that affect this area of the brain, for example, can cause "locked in" syndrome, in which the patient is fully conscious and aware, but is unresponsive, even though their muscles are not paralyzed. Such patients appear to be unconscious even though they may be fully aware, and can be mistakenly diagnosed as having coma, or even brain death.[80]

Experiments have shown that during deep, dreamless sleep, connections and relays between the thalamus and the cortex break down. In dreaming sleep (so-called REM sleep for the Rapid Eye Movements seen in dreaming subjects) cortico-thalamic connectedness looks essentially the same as the connections during full wakefulness. Intact cortico-thalamic connectedness, for all intents and purposes, indicates consciousness, whether connected or disconnected. Experiments have shown that connectivity (and therefore connected consciousness or awareness) changes *independently* from responsiveness: one can be suppressed while the other is intact. In other words, unresponsiveness can occur in conscious people, even when nothing appears to be physically preventing the person from moving. To produce unconsciousness, a drug must therefore reduce cortical-thalamic connectivity, and not merely suppress responsiveness.

GABAergic drugs like midazolam are poor at suppressing such thalamic activity (i.e. sensory input) at doses that are known to prevent spontaneous responsiveness.[81] It has been shown that many patients who receive GABAergic drugs like midazolam as part of induction of anesthesia, and who are *not paralyzed* or are tested via the IFT and still aware and sensate *may still not voluntarily move*— i.e. will appear to be asleep— even when connected consciousness is present.[82] And it is virtually certain that all of those patients will not recall or report immobility later, even if

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[80] Das JM, Anosike K, Asuncion RMD. Locked-in syndrome. StatPearls. July 5, 2021. Available at: https://www.ncbi.nlm.nih.gov/books/NBK559026/ Accessed Aug 17, 2021

[81] Mashour GA, Pryor KO. Consciousness, Memory and Anesthesia. IN: Miller's Anesthesiology 9th Ed. Gropper MA, Ed. Elsevier, Inc. Philadelphia PA. 2002. Pp 260-66

[82] Sanders RD, Tononi G, Laureys S, Sleigh JW. Unresponsiveness ≠ unconsciousness. Anesthesiology 2012; 116:946-59

they were conscious at the time, because administration of midazolam will prevent them from remembering.

Other areas of the brain, such as the amygdala, also play a role in selecting a response out of the infinite number of responses that the brain could make when a stimulus is received. Connectivity of these areas to the cortex can determine whether an "aware" brain is able to select and perform any response. It has been shown that in various states of consciousness, damage to this area (or drug effects on this area) can prevent spontaneous responses to events, while leaving the brain's ability to follow directions (so-called "goal-directed" responses) intact.

Conditions involving the failure of cortico-thalamic connections that leave a conscious person unable to move despite not being paralyzed under non-anesthesia circumstances can help us understand what that feels like. Such neurological conditions are well-described and are called "locked-in syndromes." Some forms of locked-in syndrome are produced by certain neurological diseases, particularly a special type of stroke affecting the deep brain. With locked-in syndrome, despite having intact nerves and muscles, intact sensation, and full consciousness, patients are partially or wholly unable to move.[83] Survivors frequently report terror and desperation. One patient reported being able to hear every word and understand everything around him, including the doctors telling his wife that he had a less than 2% chance of survival, but was unable to tell them he was alive, "...in my mind I was screaming 'No!"[84]

Locked-in syndrome can also occur after administration drugs. The condition consists of being awake and aware, yet unable or minimally able to initiate movement despite not being physically paralyzed. Patients described the condition as "outward calm, but inner terror." An old anesthetic technique using butyrophenones has been all but abandoned due in large part because so many patients reported nightmares, panic, and feelings of "being locked in" during induction of anesthesia.[85,86]

Consciousness to Painful Stimuli

Any discussion of whether a particular drug "can produce unconsciousness" or "induce anesthesia" is entirely nonsensical unless it defines the particular stimulus against which the drug must produce and maintain unresponsiveness. The more severe a stimulus is, the deeper the "unconscious" state has to be to maintain unresponsiveness throughout the duration of the stimulus, and the deeper the anesthetic will generally be required to be. The person who falls asleep in front of the TV set with the volume on and the lights flickering is unconscious, for example, but will instantly awake if there is a sudden loud noise from the program or change in lights from the screen. They are, in other words, not insensate. And they are instantly no longer unconscious. Touching them or speaking to them may be all that is needed to awaken them.

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[83] Patterson JR, Crabois M. Locked-in syndrome: a review of 139 cases. Stroke 1986; 17:758-64

[84] Locked-in syndrome: rare survivor Richard March recounts his ordeal. The Guardian. Aug 7 2012

[85] Rennemo F, Larsen R, Breivik H. Avoiding psychic adverse effects during induction of neurolept anaesthesia with levomepromazine. A double-blind study of levomepromazine and droperidol. Acta Anaestheesiol Scan. 1982; 26:108-11

[86] Brown E, Purdon PL, Van Dort CJ. General anesthesia and altered states of arousal: a systems neuroscience analysis. Ann Rev Neurosci 2011; 34:601-28

A true "anesthetic" will produce a state of general anesthesia, during which no stimulus will be strong enough to elicit a response if the dose is increased to a sufficient level. But with certain drugs, such as midazolam, administration of ever-increasing amounts of drug *will actually never be sufficient to anesthetize against severely painful or noxious stimuli* because the drug reaches a predetermined "ceiling effect" beyond which giving more drug does not deepen the sedation further, and cannot match the increasingly severe stimuli (see **Ceiling Effect of Benzodiazepines**, below). This is why midazolam is not classified by the FDA as a general anesthetic, cannot be used as a solo drug produce a state of general anesthesia, and was never approved for such a use. In fact, the FDA and DEA do not even classify midazolam as an anesthetic agent (which are listed as schedule "none"), but rather as a "schedule IV controlled substance with low potential for abuse."[87]

Apart from consciousness, **awareness** of an event refers to the brain's experience of, and attention to the event, whether or not it is later remembered. Awareness is thus closely married to consciousness: one cannot attend to an event that does not enter one's consciousness. ***Recall***, on the other hand, is the ability to remember an event after it has been consciously experienced.

A person can be conscious and sensate, and experience an event, even an agonizing one, without being able to recall it. But failure to remember the event is not an indication that the person was unaware and did not experience terrible pain or suffering. Most or all of our conscious experiences will not be recalled. Who of us has not driven home from a hard day at work and realized later that we don't actually recall parts of the drive? Yet we were conscious. We saw and reacted to traffic lights and cars stopping in front of us, and realized we needed to turn down certain streets. It is well known that even experiences that are especially traumatic, e.g. painful, terrifying, or psychologically traumatizing, may in fact be associated with or even cause significant lapses in memory in the absence of drugs, even though the events occurred, were experienced, and caused terrible suffering at the time.

Unfortunately, it is common for physicians, including anesthesiology experts, to conflate the terms "awareness" and "recall", and to mistakenly assert that patients who do not *recall* intraoperative events were not therefore conscious or aware of them at the time they occurred. And yet quite the opposite has been clearly shown in multiple human clinical trials to be true. Many studies and reports demonstrate that a disturbing percentage of patients are aware to various degrees of what is happening to them while under anesthesia, but simply cannot recall it later. One reason is that many drugs used in the course of anesthesia care, such as midazolam, are actually much more efficient at producing *amnesia* (memory loss) than *unconsciousness,* whether used in combination with other, enhancing medications, or alone.

Seventy-two percent of patients exhibited purposeful responsiveness during studies using the IFT to detect awareness/responsiveness. Are the other 28% of patients who did not respond under IFT

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[87] DEA Office of Diversion Control. Available at: https://web.archive.org/web/20130522180335/http://www.deadiversion.usdoj.gov/schedules/orangebook/c_cs_alpha.pdf Accessed July 9, 2018

"unaware"? ***No. Although the IFT is the best test of awareness found so far, it misses cases of awareness***—there are patients who are awake and responsive to commands on the IFT (we know this because they report accurate memories of the operating room) and yet do not move. Domino et al,[88] found in fact in which all of the cases had recall and could accurately remember their operating room experience, only 2% moved. While some of these patients had been given paralytic agents, not all had been paralyzed.

We do not yet have an explanation for the mechanism for this finding, although it is proposed that interference with signal-switching in the thalamus may be the culprit. Terror and pain are known to impair the ability of persons to understand and carry out complex tasks during traumatic events, "freezing" them from being able to respond even when they are conscious and not under the influence of drugs.

As a cardiac anesthesiologist in the 1980s and 1990s, I had personal experience with administration of high-dose benzodiazepines for cardiac anesthesiology in an estimated 750 to 1000 cases. We never used a benzodiazepine by itself, but rather combined it with high-dose, high-potency narcotics such as fentanyl. A number of patients reported events in the operating room, demonstrating awareness with recall. In one particular case, a patient was given 1 mg/ kg of valium, plus 50 mcg/kg of fentanyl[89] (equivalent to approximately 50 to 75 mg of midazolam in a 100 kg patient *plus* a high dose of a potent IV narcotic) to induce anesthesia prior to cardiac surgery. Despite these doses he was awake and sensate, and remembered the saw cutting his chest open as well as much of the rest of his surgery. Because he had been paralyzed with pancuronium bromide, a drug closely related to vecuronium bromide, there was absolutely no indication during the surgery that he was aware—he appeared simply to be sleeping (see section on vecuronium bromide) and we did not learn of his awareness until he was able to report it to us the following day. At that time he was able to recount the entire experience to us, along with his terror, and quote conversation that was going on in the operating room at the time, proving that he was indeed conscious and not hallucinating. Given the multiple reputable peer-reviewed studies of awareness in anesthetized patients using high-dose midazolam, given that my patient had received what can only be described as "massive" dose of diazepam, and based on my overall clinical experience using high-dose benzodiazepines for cardiac anesthesia, it is my opinion to a reasonable degree of medical certainty that there is no dose of benzodiazepine that will render a prisoner insensate to the painful stimuli such as would be expected during the Oklahoma lethal injection protocol.

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[88] Domino KB, Posner KL, Caplan RA. Awareness during anesthesia. A closed claims analysis. Anesthesiology 1999; 90:1053-61

[89] Because drug dosing is depending on the size of the recipient, it is nonsensical to refer to absolute drug doses when determining the clinical appropriateness and relative "size" of a given dose. A 1 mg dose may be appropriate for a 4 kg baby, for example, but have virtually no effect on adult weighing 25 times that amount, or 100 kg. The required and administered drug doses are always expressed in terms of units per kilogram of the recipient's weight, which gives an initial indication of whether the *estimated* dose needed before adjustments for other factors (e.g. age, sex, concomitant prescription or illicit drug use, etc) is appropriate. The dose of valium referred to above would be the equivalent of 100 mg in a 200 lb person, for example, which is approximately equal in potency to 50 to 75 mg of midazolam. This dose was given *in addition* to the dosing equivalent of 5000 microgram (mcg) of fentanyl in a 100 kg person, a massive dose. Fentanyl is synergistic with midazolam and further enhances its respiratory depressant effects. To put these doses in perspective, the *usual* clinical doses of these drugs in a 100 kg person for an anesthetic induction that includes fentanyl would be 1 to 5 mg of midazolam plus 150 to 250 mcg fentanyl.

C. Midazolam has a "ceiling effect" that has been demonstrated in both animal and human studies, and is below the 500 mg dose proposed in the Oklahoma Protocol. Human studies have demonstrated that the ceiling dose does not suppress conscious reactions by human subjects after administration of midazolam, and the dose does not decrease brain activity to the degree indicative of unconsciousness as measured by EEG monitors. Administration of doses of midazolam at any dose will not overcome midazolam's inability to prevent pain and suffering.

Is it possible to achieve and maintain "unawareness" by simply delivering higher and higher doses of the midazolam? The answer is unequivocally no. This is due to the pharmacologic characteristics of all benzodiazepines, including midazolam.

It only seems logical that giving more of any given drug should result in a proportionally larger clinical effect—i.e. doubling the dose will result in twice the effect. However, many drugs, including midazolam, *do not* have proportionally larger clinical effects with increasing doses, and/or have a maximum ("ceiling" dose, also called a "saturable effect") at which no further increase in clinical effect occurs no matter how much drug is given.

With midazolam, clinical effects are produced only when midazolam binds to specific receptors (GABA receptors) in the brain to produce sedation. This works in a "key-in-lock" manner: once the receptor has bound a molecule of midazolam, it does not accept more midazolam. Up to a point, the more receptors that bind to midazolam, the more sedation is produced. The ratio of drug needed to interact with receptors is 1:1—that is, each receptor has only space to bind with one molecule of midazolam, and there are a finite and relatively small number of GABA receptors in the brain in comparison to other types of receptors. Once the maximum number of GABA receptors is bound, it does not matter how much more drug is added, because there are no receptors for it to attach to. Imagine, for example, that there are 16 total receptors that can interact with midazolam. Once 16 molecules of midazolam are administered, the maximum amount of sedation for that patient will be reached, whatever that is for each individual patient. If 32 molecules of midazolam are given, the effect is no different than if 16 are given, because there will still only be 16 receptors for the midazolam to act upon and have a clinical effect. The left-over 16 midazolam molecules will simply float around in the bloodstream, doing nothing. Even administering 160 molecules, 10-times the first dose, will have no further effect, leaving 144 unbound molecules will simply float around in the bloodstream having no further effect. This phenomenon is called a "ceiling effect", or the "saturable effect", and has been clearly shown for midazolam in dogs[90,91] and humans.[92,93] In rats, in fact, it has been shown that the dose of

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[90] Hall RI, Schwiegr IM, Hug CC Jr. The anesthetic efficacy of midazolam in the enflurane-anesthetized dog. Anesthesiology 1988; 68:862-6

[91] Seddighi R. The effect of midazolam on end-tidal concentration of isoflurane necessary to prevent movements in dogs. Veterinary Anesth Analg 2011; 38:195-202

[92] Melvin MA, Johnson BH, Quasha AL, Eger EI II. Induction of anesthesia with midazolam decreases halothane MAC in humans. Anesthesiology 1982; 57:238-41

[93] Inagaki Y, Sumikawa K, Yoshiya I. Anesthetic interaction between midazolam and halothane in humans. Anesth Analg 1993; 76:613-7

midazolam *would never be high enough to ever produce surgical anesthesia.*[94] Veterinary anesthesiology specialists at the University of British Columbia state that midazolam will not produce anesthesia in a mouse even when combined with a powerful drug like ketamine stating that in rats and mice "diazepam (i.e. valium) and midazolam (i.e. versed)….are mild sedatives so used on their own will not cause unconsciousness".[95] Even combined with ketamine, it is only recommended for use "in non-invasive, non-painful procedures, such as imaging."[96]

The ceiling effect has been demonstrated in both animal and human studies. Furthermore, it has been shown in humans that the ceiling dose of midazolam does not suppress brain function sufficiently to meet criteria for unconsciousness.

The Studies:

1. **Hall et al.**,[97] performed a classic "dose finding" pharmacologic study in dogs to determine whether midazolam could be used as a solo anesthetic drug, and to correlate the dose of midazolam and serum drug levels at which total anesthesia occurred with midazolam, if at all. They performed the study in a standard fashion for the industry, putting the dogs under "total" anesthesia with enflurane (ENF), and then administering increasing doses of midazolam to see how far they could turn down the gas and still have the dog not move to the stimulus of having a clamp put on their tail (a very painful stimulus in a dog). If midazolam can perform as a so-called "complete" anesthetic (in Dr. Antognini's words), then there must, according to the definition of an anesthetic, be a dose at which midazolam alone would keep the dogs from moving, and the ENF could be turned off. In order for this to occur, the effects of midazolam would have to have a more-or-less linear relationship to the dose (the "dose-response" relationship). In a linear "dose-response" relationship, doubling the dose of midazolam would double the effect, tripling it would triple the effect, and so on, and eventually, one would be able to cut the ENF down equally each time they increased the midazolam dose the same increment. So for example, if giving 2 mg/kg of midazolam allowed you to decrease the ENF dose by 10%, then giving 4 mg would allow you to decrease the ENF dose by 20%, 3 mg would allow a 30% reduction, etc.

   But the dose-response relationship with midazolam is not linear. The dose-response relationship is in fact exponential. As the dose of midazolam was increased, each incremental dose had less and less additional effect. With each additional increment of drug, the additional effect is smaller and smaller. This exponential dose-response relationship has been proven for midazolam in multiple studies, and it is the dose-response relationship agreed upon *by virtually*

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[94] Schaffner PL, Scherschlicht R, Polc P, et al. Pharmacology of midazolam. Arzneimittel-Forschung. 1981; 31(12a):2180-201

[95] Rat and mouse anesthesia and analgesia. University of British Columbia, Vancouver BC. March 2, 2016. Available at: https://animalcare.ubc.ca/sites/default/files/documents/Guideline%20-%20Rodent%20Anesthesia%20Analgesia%20Formulary%20%282016%29.pdf Accessed Feb 8, 2022

[96] Rat and mouse anesthesia and analgesia. University of British Columbia, Vancouver BC. March 2, 2016. Available at: https://animalcare.ubc.ca/sites/default/files/documents/Guideline%20-%20Rodent%20Anesthesia%20Analgesia%20Formulary%20%282016%29.pdf Accessed Feb 8, 2022

[97] Hall RI, Schwiegr IM, Hug CC Jr. The anesthetic efficacy of midazolam in the enflurane-anesthetized dog. Anesthesiology 1988; 68:862-6

every major pharmacologic and anesthesia textbook in the western world. It is also exactly what the known key-in-lock receptor mechanism of action of midazolam would predict.

Hall found that increasing the dose of midazolam did not further reduce the need for ENF once they reached a dose of approximately 0.3 mg/kg, even though the dose was subsequently increased over 3-fold and plasma drug levels increased from 1464 ± 293 ng/ml to 9763 ±1213. ENF was still required to keep the dogs anesthetized. Total doses of midazolam administered were as high as **11 mg/kg total** in the high dose group. And they still saw no significantly greater effect. *To put this in perspective, for a 220 lb man, the high doses would equal an intravenous dose of midazolam of **1100 mg**, more than twice the dose prescribed in the Oklahoma execution protocol.*

Note that the peak drug levels in the high dose group in which the dogs were still responsive was ***over 10,000 ng/ml*, or *more than 5 times*** the average plasma levels recorded in prisoners executed with midazolam, **and *more than 4 times the blood level of midazolam measured in John Grant following his recent execution.*** (See Appendix IV) Hall's dogs received more than 11 mg/kg of midazolam and still were moving and trying to get away from the tail clamp.

Hall et al., explicitly concluded that "at least in practical terms, there appears to be a ceiling to the anesthetic effect of midazolam."

2. **Seddighi et al,**[98] did a similar, classic dose-finding pharmacologic study in dogs, using Isoflurane (ISF) instead of ENF as the "complete" anesthetic. The technique was similar, and the stimulus used to test responsiveness was an electrical stimulus (shock supplied by electrodes placed under the skin). They gave increasing doses of midazolam while trying to turn down the ISF to find the dose of midazolam at which they could turn off the ISF and still not have the dogs respond. A loading dose of midazolam was given, followed by an infusion to maintain the plasma levels at steady state throughout the experiment. Doses of midazolam given were (loading dose and infusion):[99]

   0.2 mg/kg plus infusion of 2.5 mcg/kg/min
   0.4 mg/kg plus of 5 mcg/kg/min,
   0.8 mg/kg plus 10 mcg/kg/min,
   1.6 mg/kg plus 20 mcg/kg/min,
   3.2 mg/kg plus 40 mcg/kg/min, and
   6.4 mg/kg plus 80 mcg/kg/min

   For perspective, the highest loading dose would be equivalent to administering 640 mg to a 100 kg prisoner as an intravenous injection.

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[98] Seddighi R. The effect of midazolam on end-tidal concentration of isoflurane necessary to prevent movements in dogs. Veterinary Anesth Analg 2011; 38:195-202

[99] Note that a typical loading dose in humans is 1-2.5 mg/kg (https://www.drugs.com/dosage/midazolam.html) and the typical loading dose in dogs and cats is 0.07-0.2 mg/kg. (https://www.petplace.com/article/drug-library/drug-library/library/midazolam-versed-for-dogs-and-cats/). Humans are more sensitive to the effects of midazolam than dogs and cats.

The authors found that there was a ceiling effect in the lower dosing ranges. Even increasing the dose of midazolam **10-fold** only allowed them to reduce the total ISF dose by 30% at the maximum, after which they could not decrease the ISF further (note that the dogs still required more than 70% of the full dose of ISF). At that point drug plasma concentrations were 372 ng/ml, and increasing the dose of midazolam did allow them to turn the ISF down any further—even when they gave enough midazolam to increase the plasma concentration almost 10-fold (to 3583 ng/ml) at the maximum dose their study called for. *These plasma levels were approximately twice the average blood levels seen in prisoners executed using midazolam.* (See Appendix IV) The ceiling effect (at when point no further increase in midazolam dose had effect) was seen in dogs at a dose between 0.4 mg/kg and 0.8 mg/kg (i.e. no difference was seen between .

For perspective, the ceiling effect in dogs would correspond to 80 mg in a 100 kg inmate, if they were equally sensitive to midazolam (which they are not—humans are more sensitive and reach their ceiling effect at a lower dose as the human studies show).

The authors concluded that the 10-fold increase in drug levels "did not further reduce MACnm.[100]"They further stated that "this study demonstrated a ceiling to midazolam's effect on MACnm reduction..... The ceiling effect is thought to be due to the saturation of $GABA_A$ receptors after administration of higher doses of midazolam."

These classic pharmacologic studies scientifically prove several things that are now accepted as fact in scientific circles regarding midazolam:

1) Midazolam has a ceiling effect that in dogs appears to be at a plasma drug concentration of about 372 ng/ml.

2) Contrary to Dr. Antognini's suggestion, the dose-response curve of midazolam has been proven in multiple studies to not be linear, but exponential.

3) Midazolam can never be what Dr. Antognini refers to as a "complete anesthetic", since even at *massive intravenous* doses, it did not produce unresponsiveness. Neither research group was ever able to turn off the other anesthetic and have midazolam perform as an anesthetic by itself. *Midazolam, in other words, could not perform as an anesthetic.*

Human studies also *unequivocally confirm* the above findings.

Classic dose-finding studies such as the ones described above that include the dosing ranges used in judicial executions have not been done in humans because they would be unethical: there is no medical use for such dosages, and therefore no purpose for such studies that might outweigh even small risks of

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[100] MACnm refers to the amount of ISF required to result in no movement in only 50% of the dogs. This is a standard measure of ISF effective dose.

adverse effects associated with giving the drug to human subjects. However, we don't need such studies, because studies using lower dosing ranges have been done, and unequivocally show the ceiling affect at these lower doses. Human studies show that:

1) Midazolam has a ceiling effect at doses which are lower than the execution protocol. This means that adding more drug beyond that dose will produce no further clinical effects, including further reduction of brain activity.

2) At plasma levels of midazolam associated with maximal clinical effect, both consciousness and responsiveness persist.

3) At plasma levels that are higher than plasma levels seen in lethal injection executions, consciousness and responsiveness persist.

1. **Inagaki et al.** These authors studied women undergoing hysterectomy in a classic, dose-finding study similar in design to the dog studies above. Patients were anesthetized with halothane (HAL)—what Dr. Antognini refers to as a "complete" anesthetic agent, plus nitrous oxide (N2O).

   Once stabilized, one of the above doses of midazolam was given in the following doses:
   - **None** (control group)
   - 0.1 mg/kg followed by an infusion of 1 mcg/kg/min to keep plasma drug levels at steady state
   - 0.2 mg/kg followed by an infusion of 2 mcg/kg/min
   - 0.4 mg/kg followed by an infusion of 4 mcg/kg/min

   Steady state midazolam levels achieved in the highest dose group were 598 ± 98 ng/ml.

   The stimulus that was used to measure responsiveness was surgical incision, i.e. severe pain. Responsiveness was judged as movement with incision. In the same experimental design previously carried out in dogs in the Hall and Seddighi studies, doses of HAL were adjusted in subsequent patients down to find the lowest dose of HAL that could be achieved and not have the patients respond to incision.

   Despite serum levels of midazolam of approximately 900 ng/ml the authors were unable to turn off the HAL, meaning that once again, midazolam could not replace HAL as a "total" anesthetic. **This plasma level is over 2.3 times higher than the plasma level found in Billy Ray Irick's blood after his recent execution in Tennessee using 500 mg midazolam.**

   The authors in fact explicitly state that the dose-response curve for midazolam *was exponential and not linear.* This is consistent with both the dog studies and the known key-in-lock receptor-based mechanism of action of midazolam. They further explicitly stated that the action of midazolam had a "saturated nature," or ceiling effect. While they did not identify a specific plasma level as "the ceiling", (this study was not intended to find the specific ceiling effect dose), at the highest dose in the study, 0.4 mg/kg, the dose-response curve was considerably curved and flattened, indicating they were seeing a ceiling effect at that dose. They noted that the plasma midazolam levels in dogs required to reduce MAC are about 2.5 times the levels

required to do so in humans, and this is in range with the Seddighi finding of a ceiling effect in dogs at 0.4 to 0.8 mg/kg. Furthermore, in their study, despite plasma levels of midazolam approaching 800 ng/ml, **all of the patients were still responsive.**

To put this finding in perspective, the dose at which the beginning of a ceiling effect was seen corresponds to 40 mg in a 100 kg inmate. The plasma levels found in the patients who were still moving in this study are at or above the plasma levels seen Billy Ray Irick and Donnie Johnson, prisoners recently executed in Tennessee using midazolam. Moreover, as noted in Appendix IV, the plasma levels that result from the administration of 500 mg of midazolam can vary substantially with some levels below 400 ng/ml, and variation in two recent Oklahoma executions ranging from 3200 ng/ml for Mr. Warner and 2200 ng/ml for Mr. John Grant.

2. **Glass et al.** In this study, authors were looking at effects of 4 drugs used during anesthesia on brain activity, by comparing readings from a BIS monitor (a device that uses the patient's EEG). The drugs were alfentanil, propofol, ISF, and midazolam. In order to correlate BIS readings with "unconsciousness", they carried out a standardized sedation check, in which they softly called the patient's name, then more loudly, then shook them "mildly", then squeezed their trapezius muscle (a similar stimulus to a sternal rub). These are mild stimuli, and not severely painful.[101]

   It is known now that the sedations scale and "consciousness check" used by Glass would not be sufficient to prove unconsciousness to severe pain or noxious stimulus. Kim et al.[102] has since demonstrated in a controlled study in humans that a trapezius squeeze does not approach the level of severe pain or noxious stimulus, and that the sedation scale used by Glass to determine "unconsciousness" was insufficient to show unconsciousness to severe pain. Glass therefore erroneously categorized a response to a trapezius squeeze as being evidence of unconsciousness. It is actually a sign of responsiveness to even a mild stimulus.

   - In the midazolam group, although the target plasma concentration for the study was 300 ng/ml, the measured serum concentrations of midazolam were as high as 934 ng/ml, *and yet at these drug levels all patients who received midazolam were responsive.* These drug levels are 2.4 times the level found in Billy Ray Irick's blood following his recent execution with midazolam in Tennessee. Nine out of ten midazolam patients responded to a very light stimulus--light shaking, and the remaining patient responded to trapezius squeeze. ***No midazolam patient failed to respond.***

   - These drug levels are comparable to drug levels seen Billy Ray Irick (drug level 397ng/ml), and Donnie Johnson (drug level 934 ng/ml), the prisoners most recently executed using midazolam in Tennessee.

3. **Miyake, et al.** In this study, authors compared midazolam blood concentrations with EEG (BIS) values in a double-blind study of patients undergoing general anesthesia. Patients were given

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[101] Kim TK, Niklewski PJ, Martin JF, Obara S, Egan TD. Enhancing a dsedations score to include truly noxious stimulation: the extended observer's assessment of alertness and sedation (EOAA/S). Brit J Anaesthesia 2015; 115: 569-77

[102] Kim TK, Niklewski PJ, Martin JF, Obara S, Egan TD. Enhancing a dsedations score to include truly noxious stimulation: the extended observer's assessment of alertness and sedation (EOAA/S). Brit J Anaesthesia 2015; 115: 569-77

midazolam 0.2 mg/kg (plasma levels approached approximately 400 ng/ml), and 0.3 mg/kg (plasma levels > 600 ng/ml). The authors defined loss of consciousness as nonresponsiveness to mild stimuli (calling the name and shaking the shoulders), and used the same sedations scale that Kim et al. have since shown to not reflect surgical stimulus and not be reliable in detecting unconsciousness.

In no patient in the Miyake study did BIS values fall below 60. In fact, in most patients it remained around 70 throughout the experiment.

- **Not surprisingly, one of the patients had not only consciousness, but recall** of the operating room (despite a so-called "consciousness check" defined as indicating they were "unconscious"), and their BIS values were all above 60.

- **There was no significant difference in BIS values at the higher dose compared to the lower dose of midazolam,** i.e. that a maximal effect on the EEG (the "ceiling effect" on brain activity) occurred after administration of 0.2 mg/kg. Because the next increase in dose was to 0.3 mg/kg and there was no significant difference in scores, the ceiling effect for brain activity as demonstrated by the BIS in this study was no higher than around 0.2 mg/kg dose, and may have been lower (lower doses were not tested). It certainly was no higher than 0.3 mg/kg, since they saw the ceiling effect take place in their lower dosing group.

- **For perspective, that would be equivalent to a dose of 20-30 mg in a 100 kg inmate.**

The authors themselves concluded, yet again, that these findings are consisted with studies of other benzodiazepines, and saturation of receptors---the "saturable" or "ceiling" effect—would explain these findings. They state: "**The BIS does not decrease further [below 70] even if [midazolam's] plasma concentration increases to levels higher than that required for sedation,**" meaning that increasing the dose does not further suppress the brain beyond sedation alone, even when higher doses are given. **A lack of further depression of EEG with a doubling of the dose of midazolam demonstrates that there is a ceiling effect of midazolam on brain activity levels.**

4. **Gamble et al.**[103] These researchers studied 230 healthy patients given midazolam in combination with other drugs during the induction of anesthesia. Doses given ranged from 0.15 mg/kg to 0.5 mg/kg.

   Only 60% of patients appeared to fall asleep *when they had no stimulation* with 0.5 mg/kg midazolam. In fact, 2 patients did not even appear to be very sedated. Even when the anesthetist assessed the patient as being "asleep" after midazolam administration, once moderate stimulation (tracheal intubation) or severe stimulation (surgical incision) were applied, most responded to loud sounds and exhibited purposeful movement with the onset of the paralytic agent, indicating that they were not anesthetized, were not unconscious to severe stimulation, and could feel and react to the onset of paralysis.

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[103] Gamble JAS, Kawar P, Dundee JW, et al. Evaluation of midazolam as an intravenous induction agent. Anaesthsia 1981; 36:868-73

In a second more detailed publication of the study,[104] they note that around 40% of patients in the group receiving the 0.5 mg/kg dose of midazolam *didn't fall asleep at all.* Furthermore, while increasing the dose of midazolam from 0.2 mg/kg to 0.3 mg/kg was associated with an increase in sedation scores and responsiveness, there was little difference in sedation scores and responsiveness between the group of patients that received 0.3 mg/kg and those that received 0.5 mg/kg of midazolam. These findings are similar to, and confirm the results of **Miyake** et al. and show that increased dosing beyond this range does not lead to increased clinical effect, and in this dosing range, almost half of patients were even asleep when they weren't being stimulated, let alone when they were. Gamble et al. concluded that ***"it is obvious that in unpremedicated patients midazolam in doses up to 0.5 mg/kg is unreliable as an anesthetic induction agent."***

As a reminder: 0.5 mg/kg is at the ceiling effect of midazolam on human brain activity as measured by a processed EEG monitor, as demonstrated in the **Miyake** study cited above. Raising the dose above this will not result in increased effect or sedation.

To summarize:

- Both animal and human studies have unequivocally demonstrated a **ceiling effect** to midazolam.
- The dose-response curve for midazolam is **exponential, not linear** and has been proven in multiple studies and is accepted by *all* authoritative textbooks, and Dr. Antognin's assertion that this is not so flies in the face of all reputable sources.
- No dose of midazolam can completely "replace" an anesthetic agent on a dose-finding study, and **this by definition means that midazolam is not a "complete anesthestic"**
- The ceiling level in dogs appears to be about **370ng/ml,** which corresponded to a dose of **0.8 mg/kg** in dogs.
- Dogs are less sensitive to midazolam than humans by about 2.5 times; the doses at which a ceiling effect occurs in humans would not be greater, but almost certainly be much less than the doses in dogs—it would be illogical to think otherwise.
- Human studies show that after a dose of 0.4 mg/kg of midazolam, and at plasma concentrations of approximately **900 ng/ml,** patients are still responsive to light to moderate stimuli. This drug level is at or below plasma drug levels documented in prisoners executed using midazolam, indicating that prisoners received more than the ceiling dose of midazolam, beyond which no greater clinical effect can be seen.
- Human studies show that there is no further, suppression of the EEG or BIS occurs, when the dose of midazolam is doubled from **0.2 to 0.3 mg/kg,** and therefore the ceiling effect is at or between to those doses.

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[104] Gamble JAS, Dundee JW, Kawar P. Midazolam—an alternative to thiopentone? Br J Anaesth 1980; 52:951p-952p

- At doses of **0.2 mg to 0.3 mg/kg**, around the ceiling effect of midazolam in humans, BIS values remain above 60 and do not meet the criteria for unconsciousness.

The eyewitness accounts of John Grant's execution are exactly what we would expect, given these findings. The level of midazolam in his blood was 2200 ng/ml, far above the ceiling effect of around 600 ng/ml, meaning that the "extra" 1600 ng/ml was having no further effect. At 600 ng/ml, all patients in this study were still responsive. And there is no doubt that at the higher level, John Grant was still moving, struggling to breathe, turning his head and lurching against his restraints, demonstrating that there is no use in giving more than the ceiling dose of midazolam: it will not have any more effect in preventing responses in humans than giving a 0.2 to 0.4 mg/kg dose.

D. The Oklahoma Protocol's consciousness checks will not effectively and reliably determine whether a prisoner is unconscious and insensate to the noxious stimuli of the Oklahoma Protocol.

***Even if the prisoner does not move,* consciousness checks have been scientifically proven in human clinical and research studies to be ineffective in detecting consciousness and awareness.**

After injection of midazolam, the Oklahoma protocol calls for a "consciousness check", using "medically appropriate means" to be carried out by a member of the IV Team after injection of midazolam and before injection of vecuronium bromide and potassium chloride. This check is supposed to occur 5 minutes after completion of injection.

However, there are no such "medically appropriate" tests that can reliably detect consciousness, despite what can only be called intensive attempts by researchers to find one. Common tests studied in the literature include response to eyelash stimulation, calling of the name in a soft, then loud voice, then trapezius squeeze or sternal rub. And research shows, again unequivocally, that these measures are unable to detect awareness to severe stimulus.

The Studies : Eyelash reflex and verbal commands

Using the gold standard, the IFT, for testing awareness during general anesthesia, the following authors have demonstrated that patients are conscious to painful stimuli via IFT or demonstrated recall even when they have lost the eyelash reflex and do not respond to verbal commands:

Gamble et al.[105] **(Eyelash reflex and verbal commands)** The 1980 study cited above demonstrated a lack of anesthesia in patients receiving 0.5 mg/kg midazolam, and in the study, the authors also examined whether they (the anesthesiologists) could predict that the patient would not be responsive to severe stimulus. The "consciousness checks" they used were was

[105] Gamble JAS, Dundee JW, Kawar P. Midazolam—an alternative to thiopentone? Br J Anaesth 1980; 52:951p-952p

eyelash reflex (lightly brushing the eyelashes to see if the eyelids would twitch) and **calling the patient's name and asking them to open their eyes**. They found that even when the eyelash reflex was lost, patients still responded to commands and were therefore not unconscious. They concluded that "**absence of the eyelash reflex was not a reliable end-point**".

Russell IF.[106] **(Verbal commands)** Russell tested IFT responsiveness during two different full general anesthetic regimens in 55 patients. Patients were deemed to be unconscious before surgical incision when they failed to respond during a "consciousness check" which consisted of verbal commands. Despite appearing unconscious prior to incision, 24% opened their eyes, and 56% wrinkled their brow and shook their head when given a significantly painful stimulus (surgical incision). One patient who did not respond to verbal commands and was believed to be unconscious nevertheless exhibited "vigorous, purposeful actions that threatened to interfere with the surgical field." Note that the patient was only able to move the arm that was not paralyzed.

Baker AB.[107] **(eyelash reflex, painful stimuli)** This study involved the use of diazepam—a benzodiazepine closely related to midazolam— as part of the general anesthetic induction in 400 patients. Patients received 0.5 to 1.0 mg/kg of diazepam, (which would correspond to approximately 0.5 to 0.75 mg/kg of midazolam), followed within 1 minute by the administration of a paralytic agent, suxamethonium. They authors reported that the eyelash reflex was usually not abolished, and patients could be roused from sleep by painful stimuli despite the high dose of diazepam they were given. In other words, they were not unconscious to painful stimuli at doses that would correspond to being above the ceiling effect of midazolam.

Kocaman et al.[108] **(eyelash, verbal commands)** These researchers tested awareness using the IFT during general anesthesia involving multiple different drugs. Neither loss of eyelash reflex nor loss of response to verbal commands predicted unawareness; and awareness was demonstrated in multiple patients despite BIS levels < 60.

St Pierre et al.[109] **(verbal commands)**. These researchers demonstrated awareness using the IFT in patients undergoing general anesthesia despite loss of response to verbal commands.

In summary, loss of eyelash reflex and response to voice may occur with sedation **and has no relationship to whether a person will remain asleep once painful stimulation is applied**.

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[106] Russell IF. Comparison of wakefulness with two anaesthetic regimens. Total i.v. balanced anaesthesia. Br J Anaesth 1986; 58:965-8

[107] Baker AB. Induction of anesthesia with diazepam. Anaesth 1969; 24:388-92

[108] Kocaman Akbay B, Demiraran Y, Yalcin Sezen G, et al. Use of the bispectral index to predict a positive awareness reaction to laryngeal mask airway-Fastrach insertion and intubation. Acta Anaesthesiol Scand 2007; 51:1368-72

[109] St Pierre M, Landsleitn er B, Schwilden H, et al. Awareness during laryngoscopy and intubation: quantitating incidence following induction of balanced anaesthesia with etomidate and cisatracurium as detected with the isolated forearm technique. J Clin Anesth 2000; 12:104-8

Changes in blood pressure (BP) and heart rate (HR) during surgery or lack thereof do not correlate with consciousness and awareness, and a lack of change of blood pressure or heart rate cannot be taken as an indicator of unconsciousness.

1. **Hall et al.**[110] in the dog study described above specifically commented on the fact that in dogs there was not a significant association between HR and BP, and the occurrence of response to tail clamp. They stated "..the degree of hemodynamic response to noxious stimuli in terms of changes in heart rate and blood pressure would not be predictive of somatic (i.e. movement) responses, and *could not be relied upon as an indicator of inadequate enflurane-midazolam anesthesia."* (my italics).

As with the ceiling effect dogs, this finding has been replicated multiple times in humans.

2. **St Pierre et al.**[111] Despite patients demonstrating awareness on the IFT and in patients who have explicit recall during surgery, at least 25% of patients with a positive IFT did not show any change in BP or HR despite stimulation.

3. **Flaishon et al.**[112] Demonstrated in patients under general anesthesia who were not responsive to verbal command after propofol administration, there was no statistical association between BP, HR, and return of response to verbal commands. The authors explicitly stated that hemodynamic variables such as changes in HR and BP are not related to consciousness, and cannot be used to detect consciousness, stating "current conventional clinical monitoring may result in an undetected return of consciousness in a paralyzed patient."

4. **Hilgenberg JC.**[113] This author reported recall of surgery in a heart surgery patient, during which there was no change of HR or BP to indicate she was awake. Awareness was only discovered after surgery, when the patient recounted her experience.

5. **Moerman et al.**[114] These authors examined 26 patients who had proven consciousness under general anesthesia, and found no significant differences in hemodynamic parameters when compared to the records of patients who had no recall.

6. **Domino et al.**[115] The authors studied 61 cases of recall during surgery and found that "the classic clues for light anesthesia were absent in most cases." High BP occurred in just 15%,

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[110] Hall RI, Schwiegr IM, Hug CC Jr. The anesthetic efficacy of midazolam in the enflurane-anesthetized dog. Anesthesiology 1988; 68:862-6

[111] St Pierre M, Landsleitn er B, Schwilden H, et al. Awareness during laryngoscopy and intubation: quantitating incidence following induction of balanced anaesthesia with etomidate and cisatracurium as detected with the isolated forearm technique. J Clin Anesth 2000; 12:104-8

[112] Flaishon

[113] Hildgenberg JC. Intraoperative awareness during high-dose fentanyl-oxygen anesthesia. Anesthesiology 1981; 54:341-3

[114] Moerman N, Bonke B, Oosting J. Awareness and recall during general anesthesia. Facts and feelings. Anesthesiology 1993; 79:454-64

[115] Domino KB, Posner KL, Caplan RA. Awareness during anesthesia. A closed claims analysis. Anesthesiology 1999; 90:1053-61

fast HR in just 7%, and only 1 person moved (most, but not all patients, had received paralytic agents).

In summary, changes in HR and BP after a painful stimulus, when present, may indeed signal an aware patient, *but a lack of change in HR and BP to stimulation is just as likely to be associated with awareness as not.* A failure to detect a change in these parameters therefore cannot be taken as evidence of unconsciousness.

Trapezius squeeze

Kim TK et al.[116] studied the amount of medication required to suppress a response to a trapezius squeeze (a similar stimulus to a sternal rub), and found that only "light anesthesia" was required, when compared to electrical stimulation (or severe pain/surgery). They found that the trapezius squeeze required about half of the amount of drug to suppress than general anesthesia. They concluded "a trapezius squeeze…is not sufficiently noxious to identify clinical states consistent with typical surgical anesthesia."

E. The IV Team Leader has sworn in a declaration that the proposed methodology for consciousness checks under the Oklahoma Protocol is "Verbal, Sternum rub, painful stimulation." The IV Team Leader will not be able to tell if a prisoner is conscious using such methods. It is not credible to assert that this IV Team Leader will succeed evaluating whether a prisoner is unconscious and insensate to noxious stimuli where trained anesthesiologists and researchers with years of clinical experience specific to consciousness evaluations with thousands of patients, have not been able to do so.

Experienced Anesthesiologists Are Unable to Detect Awareness Using Clinical signs, such as loss of eyelash reflex, loss of response to commands, changes in blood pressure and pulse:

Domino et al.[117] examined 61 cases of awareness while under general anesthesia, In those cases, the anesthesiologists all judged their patients to be asleep using clinical signs, but the patients later had explicit recall of their surgery.

Moerman et al.[118] Anesthesiologists, all of which had ≥ 10 years in practice, were unable to distinguish 26 patients who were aware (and had explicit, traumatic recall of their surgery) from control patients who did not report awareness by reviewing anesthetic records and looking for clinical signs of awareness.

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[116] Kim TK, Niklewski PJ, Martin JF, Obara S, Egan TD. Enhancing a dsedations score to include truly noxious stimulation: the extended observer's assessment of alertness and sedation (EOAA/S). Brit J Anaesthesia 2015; 115: 569-77

[117] Domino KB, Posner KL, Caplan RA. Awareness during anesthesia. A closed claims analysis. Anesthesiology 1999; 90:1053-61

[118] Moerman N, Bonke B, Oosting J. Awareness and recall during general anesthesia. Facts and feelings. Anesthesiology 1993; 79:454-64

During John Grant's execution, multiple eyewitnesses, including the defense's own medical expert, report that no consciousness check was done. However, moments before "unconsciousness" was declared, Mr. Grant was visibly struggling to breathe, and purposefully moving his head. There were ample signs that he was still conscious. Even if a consciousness check were done at this point on a relatively quiet inmate, the maneuvers described by the IV Team leader (verbal, sterum rub) would be insufficient to tell if the inmate were unconscious and would remain unconscious as the sensations of suffocation, drowning and the searing pain of potassium administration continued, since these maneuvers fail to detect consciousness in humans in whom they have been studied.

F. Administration of vecuronium bromide to a conscious person causes severe pain and suffering.

Vecuronium bromide, like all paralytic agents, **has no analgesic or sedative properties of its own, and does not have any effect on consciousness.** This is scientific fact. It does not relieve pain, and it does not put anyone "to sleep." From descriptions from and lawsuits by patients who have suffered anesthetic mishap in which they erroneously received paralyzing agents (awake paralysis) including vecuronium bromide without first being sedated,[119] as well as from studies in healthy volunteers who received paralyzing agents without sedation,[120] we know that administration of such an agent to an aware patient results in extreme fear and panic, sensations of suffocation, severe air hunger, and inability to signal to providers with gestures or facial expressions that they are aware and suffering. Subjects who underwent awake paralysis in a human experiment described the sensation of being "encased in a wetsuit made of lead," that was accompanied by immediate onset of distress which worsened with attempts to move.[121] Indeed, such patients are commonly described as experiencing "outward calm and inner terror;" the outward appearance of the patient is serene because the paralysis produced by vecuronium bromide and other paralytic agents does not permit movement or changes of expression that otherwise clue an observer that the patient is in extreme terror and discomfort, even though beneath the serene exterior, the patient is fully aware and in agony. Moerman et al,[122] studied 16 patients who were conscious and paralyzed during general anesthesia. Patients reported "terror", "it was like a nightmare", "panic", "pain", sensations of "choking", and a sense of "dying". The experience was so traumatic that many had flashbacks, nightmares and required therapy for what would now be recognized as post-traumatic stress disorder.

Domino et al.[123] studied 18 cases of awake paralysis. Patients reported feeling the surgery, pain, and severe panic. The experience was so terrible, that even patients who did not experience

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[119] Domino K, Posner KL, Caplan RA, Cheney FW. Awareness during anesthesia: a closed claims analysis. Anesthesiology 1999; 90:1053-61

[120] Schuller PJ, Newell S, Strickland PA, Barry JJ. Response of bispectral index to neuromuscular block in awake volunteers. Brit J Anaesth 2015:i95-i103

[121] Ibid.

[122] Moerman N, Bonke B, Oosting J. Awareness and recall during general anesthesia. Facts and feelings. Anesthesiology 1993; 79:454-64

[123] Domino KB, Posner KL, Caplan RA. Awareness during anesthesia. A closed claims analysis. Anesthesiology 1999; 90:1053-61

pain developed severe emotional distress, were traumatized, and exhibited long-term symptoms of PTSD.

G. Vecuronium bromide administration *doubles* the risk that the prisoner will be aware.

Apart from the hazards of awareness with high-dose benzodiazepines and narcotics, scientific studies have also clearly demonstrated that administration of paralytic agents such as vecuronium bromide ***doubles the incidence of recall and awareness*** when administered with benzodiazepines and other induction agents.[124,125,126] The mechanism of this phenomenon has not been determined. Thus, not only does the use of midazolam in the Oklahoma protocol fail to achieve anesthesia, the addition of the vecuronium bromide *independently increases* the chances of the prisoner being aware. This combination of drugs is *especially likely* to result in awareness if the patient is stimulated by pain, the terror of paralysis, or the air hunger associated with suffocation.

H. Vecuronium bromide paralysis will obscure signs of extreme pain and suffering during executions rather than prevent pain and suffering.

Most deaths due to drugs, toxins or disease that occur in humans and animals involve both involuntary and voluntary movements which occur as the distressed and/or dying brain reacts to its environment. Grimacing, muscle twitching, vocalization (moaning, crying out), convulsions and "agonal" respirations all frequently occur during the dying process. While some of these movements, such as muscle twitching, can be—although not necessarily are— the result of simple spinal cord reflexes as the brain dies, others are responses to air hunger, suffocation, panic, and pain and distress that lead to conscious efforts to rouse, breathe and fight.

These movements serve a critical purpose; they signal that the individual might not be unconscious and should receive other types of drugs to reduce their awareness or pain. Masking such movements is inhumane to a dying individual because it prevents recognition and treatment of his or her suffering.

For these reasons, it is unethical to euthanize a companion animal using paralytic agents. The American Association of Veterinary Medicine forbids use of paralytic agents for euthanasia of companion animals as the "death drug," even though dying movements can be extremely distressing to owners. This is because a paralytic agent may mask the animal's suffering and hide indications that other drugs are needed.[127] The Humane Society of the United States, moreover, explicitly states that, when an animal is paralyzed,

> while the animal appears to be unresponsive to sight and sound, he may still feel deep pain and may actually be experiencing fear and

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[124] Sandin RH, Endlung G, Samuelsson P, Lenmarken C. Awareness during anaesthesia: a prospective case study. Lancet 1000; 355:707-11

[125] Bischoff P, Rundshagen I. Awareness under general anesthesia. Dtsch Arztebl Int 2011; 108:1-7

[126] Domino KB, Posner KL, Caplan RA, Cheney FW. Awareness during anesthesia: a closed claims analysis. Anesthesiology 1999; 90: 1053-61

[127] Leary S, Underwood W, Anthony R, et al. American Veterinary Medical Association Guidelines for the euthanasia of animals: 2013 edition. AVMA, Schaumburg Il.

> panic as he remains aware of his surroundings. For this reason, immobilizing agents are never appropriate for use in euthanasia.[128]

In the case of medical care for terminally ill human beings, it would be unthinkable as well as unethical to remove a patient from a ventilator, and then for appearances sake, paralyze them and leave them to suffocate in a lingering death—aware of what was happening to them, but unable to cry out for help. Professional guidelines in human medicine explicitly oppose such practice and state that it is both unethical and inhumane because it would mask signs of suffering that need treatment.[129,130]

I. Injection of Potassium Chloride into a vein causes severe and excruciating pain, like "being burned alive."

Potassium chloride (or KCl) is a naturally occurring mineral salt that can be administered orally or intravenously. Potassium ions perform a number of functions in the human body, many of which involve activation of channels in cells that regulate the flow of substances in and out of the cells, and conduct of electrical energy along nerves, muscles and in the heart. Rapid IV administration of potassium chloride is used in lethal injection protocols to cause rapid onset of a fatal heart arrhythmia, i.e., to "stop the heart."

Injection of undiluted potassium chloride (e.g. 2 mEq/ml) causes extreme and excruciating pain, and therefore in medical practice is only given in very dilute concentrations, and very slowly. Potassium chloride is never intentionally given "IV push," (i.e. as a bolus in a relatively small [< 100 ml] volume of fluid), even in total doses of 25 mEq. This is because even such a small dose—which is not likely to produce heart arrhythmias—when diluted in small volumes of fluid (< 1 liter), results in a high enough concentration of potassium chloride that the drug cauterizes or "sears" the tissues lining the veins when it comes in contact with them, rupturing cells, destroying tissues in and around the vein and causing agonizing pain. Concentrations of more than 80-100 mEq/L (milliequivalents per liter)[131] are known to cause *excruciating* pain, sometimes described by patients as if someone poured gasoline on their arm and lit it with a match, or applied a blowtorch to their skin.[132] This pain does not stop after injection is completed, because the injury to the lining of the blood vessels continues to grow.

I have personally heard a patient scream when a concentration of 40 mEq diluted in 100ml (a concentration of 400 mEq/L and only about 1/5th the concentration used in the Oklahoma protocol and

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[128] The Humane Society of the United States. Euthanasia Reference Manual, 2013. Available at: https://www.animalsheltering.org/sites/default/files/content/euthanasia-reference-manual.pdf Accessed Nov 2, 2017

[129] Downar J, Delaney JW, Hawryluck L, Kenny L. Guidelines for the withdrawal of life-sustaining measures. Intens Care Med 2016; 42:1003-17

[130] Truog RD, Campbell ML, Curtis JR, et al. Recommendations for end-of-life care in the intensive care unit: A consensus statement by the American College of Critical Care Medicine. Crit Care Med 2008; 36:953-63

[131] The convention for dosage of mineral salts such as potassium chloride is in mEq, or in mEq per liter of whatever fluid in which they are diluted. A physician must always not only designate the total dose (mEq), but how to dilute the salt (mEq/L) because both affect the clinical effects. For context, a common dose range of potassium chloride is 25 to 40 mEq, delivered in 1 liter of fluid (i.e. a concentration of 25 to 40 mEq/L) over 8 to 10 hours. In the Oklahoma protocol, the potassium chloride is undiluted, and given as a concentration of 2 mEq/cc, the equivalent of 2000 mEq/L—a concentration that is 50 times greater than that known to cause humans to scream during injection.

[132] Personal communications by patients to Expert during quality insurance investigations of anesthetic mishaps 1994-2000.

more than 20 times as concentrated as doses known to cause severe pain) was inadvertently administered too rapidly.[133]

Given the fact that midazolam has no analgesic properties, and has been shown to be associated with a high probability of awareness in the operating room (see below), and given that the anticipated dose and concentration of potassium chloride to be administered in the Oklahoma protocol is certain to cause excruciating pain during injection, it is my expert opinion that the prisoner will certainly be aware of the injection and will experience excruciating pain. Additionally, because of the administration of vecuronium bromide prior to the injection of potassium chloride, (which further enhances this likelihood), it is my expert opinion that it is also very likely that during most executions under this protocol observers will not see any movements from the awake prisoner that they will recognize as indicating the prisoner is aware and in pain.

Summary Opinion

In summary, it is my expert opinion the Oklahoma protocol is certain to result in a sensate prisoner being aware of severe pain and noxious stimuli, such as sensations of drowning, suffocation, terror, and searing pain with injection of potassium chloride, even while there may be few outward signs of pain and suffering due to effects of midazolam and or simultaneous paralysis by vecuronium.

Signed: Mark A. Van Norman MD

Dated: 2/4/2022

[133] Pucino F, Danielson BD, Carlson JD. Patient tolerance to intravenous potassium chloride with and without lidocaine. Drug Intell Clin Pharm 1988; 22:676-9

Appendix I: Gail A. Van Norman MD Curriculum Vitae

CURRICULUM VITAE
Gail A. Van Norman, M.D.

Education:

1973-1977	University of Washington, Seattle, Washington	Honors B.S, Microbiology
1977-1981	University of Washington School of Medicine, Seattle, Washington	M.D. with Honors

Postgraduate Training:

1981-1982	Virginia Mason Hospital, Seattle	Internship	Internal Medicine Washington
1982-1984	Virginia Mason Hospital, Seattle, Washington	Residency	Internal Medicine
1986-1988	University of Washington, Seattle, Washington	Residency	Anesthesiology
1988-1989	University of Washington, Seattle, Washington	Fellowship	Cardiothoracic Anesthesiology
1992-1993	University of Washington, Seattle, Washington, Department of Biomedical Ethics	Certification	Health Care Ethics
2001	Perioperative Transesophageal Echocardiography Examination	Testamur	
2011	ASA Business Management Certification	Certification	

Faculty Positions Held:

1989-1994	Clinical Acting Instructor, Department of Anesthesiology, University of Washington, Seattle, Washington
1994-1995	Acting Instructor, Department of Anesthesiology, University of Washington, Seattle, Washington
1995-1997	Acting Assistant Professor, Department of Anesthesiology, University of Washington, Seattle, Washington
1997-2000	Assistant Professor, Department of Anesthesiology, University of Washington, Seattle, Washington
1997-2000	Adjunct Assistant Professor, Department of Internal Medicine, University of Washington, Seattle, Washington
2000-2001	Clinical Assistant Professor, Department of Anesthesiology, University of Washington, Seattle, Washington
2001 -2008	Clinical Associate Professor, Department of Anesthesiology, University of Washington, Seattle, Washington
2008-	Professor, Department of Anesthesiology, University of Washington, Seattle, Washington
2008-	Adjunct Professor, Department of Biomedical History and Ethics, University of Washington, Seattle, Washington

Hospital Positions Held:

1984-1985	Attending Internist, Jefferson Memorial Hospital, Port Townsend, Washington
1985-1986	Attending Internist, Highline Community Hospital, Burien, Washington
1989-1992	Staff Anesthesiologist, Northwest Hospital, Seattle, Washington
1992-1994	Staff Anesthesiologist, Swedish Hospital, Seattle, Washington
2000 – 2008	Staff Anesthesiologist, St. Joseph Medical Center, Tacoma, Washington
2000 – 2006	Director, Transesophageal Echocardiography Education, Department of Anesthesiology, St. Joseph Medical Center, Tacoma, Washington
2003-2004	Clinical Director, Department of Anesthesiology, St. Joseph Medical Center, Tacoma, Washington
2008-2013	Medical Director, PreAnesthesia Clinic, University of Washington Medical Center, Seattle, Washington
2010-	Physician Champion, Compliance Officer, Dept of Anesthesiology and Pain Medicine, University of Washington, Seattle WA

Non-Hospital Positions Held:

1984-1985	Consulting Internist, Spokane Urban Indian Health Center, Spokane, Washington
1985-1986	Consulting Internist, Seattle Community Health Clinics serving economically disadvantaged patients; for Group Health Cooperative of Puget Sound, Seattle, Washington
2005-2006	Chair CQI Process, Pacific Anesthesia, Inc., Tacoma, Washington
2006 -2008	Board of Directors, Pacific Anesthesia, Inc., Tacoma, Washington
2007-2008	Vice President, Pacific Anesthesia, Inc., Bellevue, Washington

Honors:

1978	Medical-Scientist Traineeship Grant, University of Washington, Seattle, Washington
1980	Alpha Omega Alpha
1981	Merck Manual Medicine Award
1981	John J. Bonica Anesthesiology Award, Department of Anesthesiology, University of Washington
1985	Award of Merit for Service to the Health Care Needs of Native Americans, Spokane Urban Indian Health Service
2000	President's Award, Pacific Anesthesia, Inc. Tacoma, Washington
2008	Mary Jane Kugel Award, Medical Science Review Committee, Juvenile Diabetes Research Foundation International
2011	Brocher Foundation Residency in Ethics

Board Certification:

1984	American Board of Internal Medicine
1990	American Board of Anesthesiology

License to Practice:

1981-	Washington State

1991-2003 Wisconsin

Professional Organizations:

1984-1987 American Society of Internal Medicine
1989-2000 King County Medical Society
1989-2000 Washington State Society of Anesthesiologists; Co-chair, Medical Education Committee, 1994-1997
1989-2013 American Society of Anesthesiologists; Committee on Ethics, 1992-2013
2003-2007 Society of Cardiovascular Anesthesiologists; Committee on Ethics; 2003-2007
2008-2013 American Society of Bioethics and Humanities
2015-present International Academy of Law and Mental Health
2015-present Overseas Fellow, Royal Society of Medicine

Teaching Responsibilities:

Lectures:

Undergraduate Student Lectures:

1999-2008 University of Washington, Undergraduate Introduction to Bioethics Course (MHE 411) "Informed Consent"
2001 University of Washington, Seattle Biomedical Ethics for Medical Students Lecture Series, "Informed Consent"
2008 University of Washington Dept. of Biomedical History and Ethics: MHE 597C, Informed Consent in Clinical Practice
2009-2017 University of Washington Dept. of Biomedical History and Ethics: MHE 597C, Informed Consent

Resident Lectures:

1992-1994 University of Washington, Department of Anesthesiology, "Clinical Ethical Issues in Anesthesia Practice"
1994-2000 University of Washington, Department of Anesthesiology, Resident Core Lecture series, "Perioperative Diabetes Management"
1994-2000 University of Washington, Department of Anesthesiology, Resident Core Lecture Series "Anesthetic Implications of Neuromuscular Disease"
1994-2000 University of Washington, Department of Anesthesiology, Resident Core Lecture Series, "Pathophysiology of Ischemic Heart Disease"
1994-2000 University of Washington, Department of Anesthesiology, Resident Core Lecture Series, "Intraoperative Management of the Patient with Ischemic Heart Disease"
1994-2000 University of Washington, Department of Anesthesiology Resident Core Lecture Series, "Preoperative Evaluation of the Patient for Anesthesia and Surgery"
1994-2000 University of Washington, Department of Anesthesiology, Resident Core Lecture Series, "CQI: Quality Improvement in Practice"
1994-2000 University of Washington, Department of Anesthesiology, Resident Core Lecture Series, "Post Operative Cognitive Dysfunction"
1994- University of Washington, Department of Anesthesiology, Resident Core Lecture Series, "Clinical Ethical Issues in the Practice of Anesthesiology,"
1994- University of Washington, Department of Anesthesiology R2 Core Lecture series, "Ethical Issues of Informed Consent"

1994-	University of Washington, Department of Anesthesiology R2 Core Lecture series, "Do Not Resuscitate Orders in the Operating Room"
1994-	University of Washington, Department of Anesthesiology R3 Core Lecture series, "Ethics of Surrogate Consent"
1994-	University of Washington, Department of Anesthesiology R3 Core Lecture series, "Ethical Issues in Organ Transplantation"
1994-	University of Washington, Department of Anesthesiology R4 Core Lecture series, R4 Seminar: "Allocation of Scarce Resources in a Managed Care Environment"
1995	University of Washington, Department of Anesthesiology, Resident Special Evening Lecture Series: Forum on Ethical Issues in Anesthesiology, "Informed Consent and Surrogate Consent: Who Speaks for the Patient?"
1995	University of Washington, Department of History and Ethics, Ethics Brown Bag Lecture Series, "Ethical Dilemmas in the Operating Room"
1995, 1997	University of Washington Department of Anesthesiology, Evening Resident Special Workshop, " Fiberoptic Intubation and Management of the Difficult Airway"
1996	University of Washington, Department of Anesthesiology, Resident Special Evening Lecture Series: Forum on Ethical Issues in Anesthesiology, "Ethical Issues in Organ Transplantation, and The Impaired Practitioner"
1997	University of Washington, Department of Anesthesiology, Resident Special Evening Lecture Series: Forum on Ethical Issues in Anesthesiology, "Physician-Assisted Suicide, and the Impaired Physician,"
1997	University of Washington, Department of History and Ethics; Ethics Brown Bag Lecture Series "DNR in the Operating Room: Should Different Rules Apply?"
1997	University of Washington, Department of History and Ethics, Ethics Brown Bag Lecture Series "Ethical Pain Management in the Addicted Patient Undergoing Surgery--Is There A Duty to Rescue?"
1999	University of Washington, Department of Biomedical Ethics and History, Master's Course in Biomedical Ethics, "Informed Consent."
1999	University of Washington, Department of Biomedical Ethics and History Ethics, Brown Bag Lecture Series, "Who is Captain of the Ship on the Multidisciplinary Team: Lessons from the Operating Room"
2004	University of Washington, Department of Anesthesiology, Resident Special Evening Lecture Series: Forum on Ethical Issues in Anesthesiology "Ethics of Organ Transplantation"
2008-	University of Washington, R2 Core lecture "Introduction to Preoperative Evaluation."
2008-present	University of Washington CA1 PAC lecture series: "informed Consent/Informed Refusal"
2010-present	University of Washington Resident Introductory Lecture series: "EHR Integrity"
2010-present	University of Washington R2 and R3 Core Lectures: "Coding and Documentation"
2021-present	University of Washington Resident Core Lectures, CA1-3, Ethical Responsibilities of Anesthesiologists

Grand Rounds Lectures

1994-1996	New England Deaconess Hospital, Boston, Massachusetts: "Ethical Issues in Anesthesia Practice"
1994	University of Washington, Ethics Grand Rounds, "DNAR in the Operating Room"
1996	University of Washington, Hematology Grand Rounds, "Antifibrinolytics, Use, Clinical Efficacy, and Cost Effectiveness"
1998	Providence Medical Center, Department of Surgery, Surgery Grand Rounds "Ethical Issues in Surgical Care: A Panel Discussion"
1998	University of Washington School of Medicine: Combined Cardiothoracic Surgery/Cardiology Grand Rounds, "Brain Death and Organ Donation"

2001	Rush-Presbyterian-St. Luke's Medical Center, Anesthesia Grand Rounds, Department of Anesthesiology Chicago, Illinois, "Historical Perspectives on the Ethics of Clinical Research"
2002	University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, Department of Anesthesiology, Anesthesia Grand Rounds, "Ethical Issues in Brain Death"
2008	University of Washington Anesthesiology Grand Rounds: "Perioperative Management of Pacers and AICDs"
2009	University of Washington Medical Center Combined Anesthesiology and Surgery Grand Rounds "Preoperative Evaluation: Where Have We Been, Where are We Going?"
2009	University of Oklahoma Anesthesiology Grand Rounds, "Is a Free Pen Just a Free Pen? Conflicts of Interest in Clinical Practice, Research and Industry." February, 2009.
2009	University of Washington Department of Anesthesiology; DNR in the Operating Room April, 2009
2010	University of Washington Department of Orthopedics; Preoperative Testing: Should Your Patient have a Preoperative Chest XRay? April, 2010.
2010	University of Washington Department of Obstetrics and Gynecology: Preoperative Testing: Less is More. May, 2010
2011	MD Anderson Cancer Center; Houston Texas. Dept of Anesthesiology. Fraud and Plagiarism in Medical Research. February 14, 2011
2012	MD Anderson Cancer Center: Houston Texas. Dept of Anesthesiology. Physician Assisted Suicide and Euthanasia. April 18, 2012
2012	MD Anderson Cancer Center, Houston Texas. Risk Management Department. Fraud and Plagiarism in Medical Research. April 18 2012
2012	Overlake Medical Center Department of Anesthesiology: Lifeboat Ethics. April, 2012 Bellevue, WA
2018	University of Washington "Respecting Patient Privacy". Feb 28, 2018
2021	University of Washington. Compliance and Key Documentations Changes in EPIC. January 20, 2021

Resident Journal Club:

1995	University of Washington, Department of Anesthesiology "Use of Magnesium for Cardiac Surgery Patients"
1996	University of Washington, Department of Anesthesiology; "Anesthesia for IVF, Teratogenicity of Anesthetics, and Anesthesia and the Breast-Feeding Patient"
1996	University of Washington, Department of Anesthesiology, "N2O: Friend or Foe?"

Faculty Lectures:

1995	University of Washington, Department of Anesthesiology, CME lecture, "Ethics and the Examiners"

Workshops:

1994-1996	University of Washington, Department of Anesthesiology, CME Workshop, "Difficult Airway"
1996	University of Washington School of Medicine CME Workshop, "Aprotinin and Other Antifibrinolytics," Blood Therapy: Applications and Alternatives"

Nursing Lectures:

1996-1997	University of Washington Medical Center, Pre-Surgical Clinic Nursing Lecture Series; "Preoperative Assessment of the Patient for Anesthesia"
1999	University of Washington Medical Center Operating Room, Nurses Weekly Conference, "Informed Consent in the Operating Room."
1999	University of Washington, Association of Operating Room Nurses, Perioperative Nursing Internship Program "Informed Consent in the Operating Room"
2000	University of Washington, Department of Radiology Nursing Staff Lectures, "Sedation of the Patient with Severe Liver Disease for TIPS Procedure"
2008	University of Washington Medical Center Operating Room Nursing Conference, "Informed Consent in the Operating Room."
2011	Overlake Medical Center, Bellevue WA. Perioperative Nursing Education. DNR in the OR.
2020-present	PreAnesthesia Clinic Daily Huddle "Covid Moment"—developments in covid pandemic, including viral behavior, vaccine development and variants of the virus.

Editorial Responsibilities:

1997-2003	Consulting Editor, ASA Syllabus on Ethics, American Society of Anesthesiologists, Park Ridge, Illinois
2004-	Editor, ASA Syllabus on Ethics, American Society of Anesthesiologists, Park Ridge, Illinois.
2009	Editor-in-Chief, *Clinical Ethics for Anesthesiologists, a Cambridge University Press Case-Based Textbook.*
2012-2017	Associated Editor, North America Clinical Ethics, Journal of Bioethical Inquiry.
2019	Editorial Board Member, ClinicalKey Procedures, Elsevier Inc, Philadelphia PA
2021	Editorial Board Member, ClinicalKey Clinical Reviews, Elsevier Inc, Philadelphia PA
2020-2021	Invited Editor, Journal of Bioethical Inquiry, Springer, Australia

Special National/International Responsibilities:

1992-2014	Committee on Ethics, American Society of Anesthesiologists, Park Ridge, Illinois
1994	Panelist, American Society of Anesthesiologists Annual Meeting, Panel on Ethics: Ethical Issues in Anesthesiology
1995	Moderator Ethics Panel, American Society of Anesthesiologists, Annual Meeting, "Is There a Role for the Anesthesiologist in Physician-Assisted Suicide?"
1996-2006	Moderator Clinical Forum, American Society of Anesthesiologists, Annual Meeting, "Ethics/Geriatrics"
1996-1999	Moderator Problem-Based Learning Discussion, American Society of Anesthesiologists Annual Meeting, Ethics cases
1997	Panelist, American Society of Anesthesiologists Annual Meeting Panel on Education, "Can Ethics be Taught?"
1998	Workshop Organizer and Lecturer, American Society of Anesthesiologists, "Teaching Clinical Ethics in Anesthesia Residency"
1999	Invited Participant, Duke University, Durham, North Carolina, Second Duke Conference on Surgery and the Elderly

2001	Panelist, American Society of Anesthesiologists Annual Meeting Panel on Professionalism, "Defining and Demanding Excellence in Anesthesia Job Performance"
2004	Panelist International Liver Transplantation Society Annual Meeting, "Ethics of Liver Transplantation, NHB/DCD Donors: Perioperative Issues in Liver Transplantation"
2005	Representative for the American Society of Anesthesiologists (one of four), First National (UNOS) Conference on Donation After Cardiac Death, Philadelphia [please see Bernat J.L. et al. Report of a National Conference on Donation After Cardiac Death. Am J Transpl 6: 281-91, 2006.]
2005-	Ethics Reviewer, Research and Funding Department, Juvenile Diabetes Research Foundation, New York
2006	Panelist, American Society of Anesthesiologists Annual Meeting, Panel on Professionalism, "Role of the Anesthesiologist in End-of-Life Care –Do physicians have conflicts of interest?"
2006	Panel Moderator, American Society of Anesthesiologists Annual Meeting, Panel on Professionalism, "Working Hard—or Sleeping at the Wheel. Should the ASA adopt aviation-style standards for work hours for anesthesiologists?"
2006	Panel Moderator, American Society of Anesthesiologists Annual Meeting: Panel on Ethics, "Should Anesthesiologists Participate in Executions?"
2006-2008	Ethicist, Medical Science Review Committee, Juvenile Diabetes Research Foundation International.
2007	Panelist, ASA Panel on Ethical Issues in Perioperative Medicine
2007	Panelist, ASA Panel on Professionalism in Multidisciplinary Teams: Palliative Care and Multidisciplinary Pain Management
2007	Panelist, ASA Panel "What is Professionalism? Do I Need it? Do I Have it?"
2007	ASA Clinical Forum: Special Topics in Bioethics
2008-2011	Chair, American Society of Anesthesiologists Committee on Ethics
2008	Panelist, ASA Panel "Lethal Injection."
2008	Panelist, ASA Panel "DCD—do we need it? Con."
2008	Moderator, ASBH panel "Opioid Pain Medication for Chronic Nonmalignant Pain: A Right or a Wrong?" American Society of Bioethics and Humanities
2008	Invited lecturer: 37th Annual Advances in Family Practice and Primary Care, August. Seattle WA: "DNR and Other Advance Directives in the OR"
2008	Invited Lecturer: AANA. "Preoperative Testing" and "Perioperative beta blockade." September. Spokane, WA
2009	Refresher Course Lecture: Ethics for Anesthesiologists in the 21st Century. New Orleans LA.
2010	Refresher Course Lecture: Protecting Vulnerable Subjects in Research: Ethical Obligations to Human and Animal Research Subjects. San Diego, CA
2010	Panelist, ASA Panel "Health Care Reform." ASA Annual Meeting, San Diego, CA.
2011	Invited lecturer: "DNR Orders in the Perioperative Period." Overlake Medical Center, Bellevue Washington.
2011	Should Anesthesiologists Participate in Physician-Assisted Suicide? Pro and Con. American Society of Anesthesiologists Annual Meeting. Chicago, Il. 2011
2012	Invited Lecturer: Informed Consent and Informed Refusal in the OR. Puget Sound Multi-Chapter AORN Coalition. Kent, WA Jan 2012
2012	Invited lecturer, American Society of Interventional Pain Physicians Refresher Course and Review. Fraud in Anesthesia Research, and Ethics of Interventional Pain Management. April 2012 Phoenix, AZ
2012	Invited lecturer, American Society of Interventional Pain Physicians Refresher Course and Review. Fraud in Anesthesia Research, and Ethics of Interventional Pain Management. August 2012 San Francisco, AZ

2012	Invited Lecturer: 41st Annual Refresher Course for Nurse Anesthetists. Ethics of Informed Consent; Ethics of Informed Refusal; Ethical Issues in Preoperative Testing. Nov 2012 Orlando, FL.
2012	Invited Lecturer: Idaho State Society of Anesthesiologists: The Ethics of Preoperative Testing. Boise, Idaho, Spring 2012.
2013	Invited lecturer, American Society of Interventional Pain Physicians Refresher Course and Review. Ethics of Interventional Pain Management. February 2012 Phoenix, AZ
2013	Invited lecturer, American Society of Interventional Pain Physicians Refresher Course and Review. Ethics of Interventional Pain Management. October 2013 Denver, CO
2013	Panelist: Controversial Cases in Organ Donation and End-of-Life Care, American Society of Anesthesiologists Annual Meeting, San Francisco CA.
2014	Panelist: Controversies in Organ Transplantation, American Society of Anesthesiologists Annual Meeting, New Orleans LA
2014	Ethical Issues Regarding Open Access Journals, American Society of Bioethics and Humanities Annual Meeting, San Diego CA.
2015	Harvard Anesthesia Update 2015. Point/Counterpoint. Physician Involvement in Lethal Injection. May 2015
2015	Invited lecturer, American Society of Interventional Pain Physicians Refresher Course and Review. Ethics of Interventional Pain Management. Chicago Il, July 2015
2015	Faculty, Moya Annual CRNA Refresher Course. Orlando, FA. Nov 2015
2020	Invited Panelist, When does the anesthesiologist get to decide? Conscientious Objection: What it Is—and What it Isn't. American Society of Anesthesiologists Annual Meeting. (held virtually due to COVID 19)

Special Regional Responsibilities:

1991-1993	Member, Medical Ethics and Practice Committee, King County Medical Society, Seattle, Washington
1992-1994	Member, Professional Liability Panel, King County Medical Society, Seattle, Washington
1992-1996	Co-chair, Committee on Education, Washington State Society of Anesthesiologists, Seattle, Washington
1995	Program Chair, Washington State Society of Anesthesiologists Spring Meeting, "Controlling Our Destiny: Leadership Opportunities Beyond the Operating Room"
1995	Program Chair, Washington State Society of Anesthesiologists Fall Meeting, "The Difficult Airway: Clinical Approaches and Risk Management"
1996	Program Co-Chair, Washington State Society of Anesthesiologists Spring Meeting, "Preoperative Issues for the Surgical Patient"
1996	Representative, Washington State Society of Anesthesiologists, ASA Legislative Session, Washington, DC
2021	Group/Discussion leader for University of Washington Department of Bioethics and Humanity's annual Summer Seminar in Healthcare Ethics. Seattle WA. August 2021

Special Local Responsibilities:

1991-1993	Ethics Committee, Swedish Hospital, Seattle, Washington
1995-2000	Associate Medical Director, Pre-surgery Clinic, University of Washington Medical Center, Seattle, Washington
1996-1997	Member, Advisory Committee on Ethics, University of Washington Medical Center, Seattle, Washington

1998-2000	Chair, Continual Quality Improvement University of Washington Medical Center, Department of Anesthesiology
1997-2000	Co-chair, Advisory Committee on Ethics, University of Washington Medical Center, Seattle, Washington
1999-2000	Acting Chief, Cardiothoracic Anesthesia, University of Washington Department of Anesthesiology
2006 -	Regional Ethics Committee member, Franciscan Health Care Systems, Tacoma, Washington
2006-2008	Member, Regional Committee on Organ Transplantation, Franciscan Health Care System, Tacoma, Washington
2008-2009	Member, Joint Transfusion Committee, HMC and UWMC
2009-2010	Member, Standards and Finance Committee, University of Washington Department of Anesthesiology and Pain Medicine
2010-2016	Member, Business Excellence Committee, University of Washington Physicians
2010-	Chair/co-chair Standards and Finance Committee, University of Washington Department of Anesthesiology and Pain Medicine
2010-present	Compliance Officer, Department of Anesthesiology and Pain Medicine, University of Washington, Seattle WA.
2014-present	Member, Dept of Anesthesiology and Pain Medicine Promotions Committee

Research Funding:

University of Washington Department of Anesthesiology; "The Effects of PTCA vs. CABG in Reducing Postoperative Cardiac Morbidity in Patients Undergoing Noncardiac Surgery"; 1995; $500 [Investigators: Chan V, Van Norman G, Posner K]

Washington State Society of Anesthesiologists; The Effects of PTCA vs. CABG in Reducing Postoperative Cardiac Morbidity in Patients Undergoing Noncardiac Surgery; 1995; $2000; [Investigators: Chan V, Van Norman G, Posner K]

Washington State Society of Anesthesiologists: Echocardiography Screening in the PreAnesthesia Clinic. 2010. $5000 [Investigators: Wako E, Van Norman GA, Rooke A, Otto C.]

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Publications in Refereed Journals or Websites:

1. Van Norman G., Groman N.. A Method of Quantitating Sensitivity to a Staphylococcal Bacteriocin. Infection and Immunity 26(2): 787-789, 1979.

2. Dreis D., Winterbauer R., Van Norman G, Sullivan S., Hammer S. Cephalosporin-Induced Interstitial Pneumonitis. Chest 86(1): 138-140, 1984.

3. Winterbauer R., Hammer S., Van Norman G. Histiocytosis X, Case Report and Discussion. J Resp Dis February 1985.

4. Van Norman G, Pavlin E, Eddy C, Pavlin J. Hemodynamic and Metabolic Effects of Aortic Unclamping Following Emergency Surgery for Traumatic Thoracic Aortic Tear in Shunted and Unshunted Patients. J Trauma 31(7): 1007-1016, 1991.

5. Van Norman, G. Diabetes in the Operating Room: Metabolic Challenges for the Anesthesiologist. Seminars Anesth 14(3): 210-220,1995

6. Van Norman, G. Preoperative Assessment of Common Diseases in the Outpatient Setting. Anesthesiology Clinics of North America. 14(4): 631-654,1996.

7. Van Norman G. Preoperative Management of Common Minor Medical Issues in the Outpatient Setting. Anesthesiology Clinics of North America. 14(4): 655-678, 1996.

8. Aziz S, Haigh W, Van Norman G, Kenney R, Kenney M. Blood Ionized Magnesium Concentration During Cardiopulmonary Bypass and Their Correlation with Other Circulating Cations. J Card Surg Sep-Oct 11(5): 341-7, 1996.

9. Van Norman G, Gernsheimer T, Chandler W, Cochran P, and Spiess B. Indicators of Fibrinolysis During Cardiopulmonary Bypass After Exogenous Antithrombin III Administration for Acquired Antithrombin III Deficiency. J. Cardiothoracic Vasc Anesth 11(6): 760-763, 1997.

10. Jackson S, Palmer S, Van Norman G, et al. Ethical Issues in Anesthesia. Adv Anesth 14:227-260, 1997.

11. Van Norman G. A Matter of Life and Death: What every anesthesiologist should know about the medical, legal, and ethical aspects of declaring brain death. Anesthesiology 91(1): 275-287, 1999.

12. Posner K, Van Norman G, Chan V. Adverse Cardiac Events Following Noncardiac Surgery in Patients with Coronary Artery Disease Undergoing Prophylactic PTCA. Anesth Analg 89: 553-60, 1999.

13. Reilly DF, McNeely JM, Doerner D, Greenberg DL, Staiger TO, Geist MJ, Vedovatti PA, Coffey JE, Mora MW, Johnson TR, Guray Ed, Van Norman GA, Fihn S. Self-Reported Exercise Tolerance and the Risk of Serious Perioperative Complications. Arch Int Med 159: 2185-92,1999.

14. Van Norman G. Ethics and The Elderly Patient. Current Anesth Rep 1:13-17, 1999.

15. Pavlin DJ, Arends RH, Gunn H, Van Norman G, Keorschgen M, Shen D. Optimal Propofol-Alfentanil Combinations for Supplementing Nitrous Oxide for Outpatient Surgery. Anesthesiology 91(1): 97-108, 1999.

16. Jackson S, Van Norman, G. Goals and Values Directed Approach to Informed Consent in the "DNR" Patient Presenting for Surgery--More Demanding of the Anesthesiologist? Editorial. Anesthesiology 90:3-6, 1999.

17. Pavlin JD, Colley PS, Weymuller EA, Van Norman GA, Gunn HC and Koerschgen M. Propofol Versus Isoflurane For Endoscopic Sinus Surgery. Am J Otolaryn; 10: 96-101, 1999.

18. Van Norman, G. Angioplasty and Noncardiac Surgery: Risks of Myocardial Infarction. Current Opinion in Anesthesiology, 12(1):15-20, 1999.

19. Van Norman, G. Response: Re: Can Brain Death Testing Be Perfect? Anesthesiology. 92(4): 1204-5, 2000.

20. Van Norman G, Patel MA, Robledo J, Chandler W, and Vocelka C. Effect of Hemofiltration on Serum Aprotinin Levels in Patients Undergoing Cardiopulmonary Bypass. J. Cardiothor Vasc Anesth 14(3): 253-256, 2000.

21. Van Norman G, Palmer S. Coercion and Restraint in Anesthesia Practice. International Clinics of Anesthesiology. Medical Ethics 39(3): 131-143, 2001.

22. Van Norman, G. Ethical Issues and the Role of Anesthesiologists in Non-Heart-Beating Organ Donation. Current Opinion Anesthesiology 16(2): 215-9, 2003

23. Van Norman G. Another Matter of Life and Death; What Every Anesthesiologist Should Know About the Ethical, Legal and Policy Implications of the Non-Heart-Beating Organ Donor. Anesthesiology 2003. 98(3):763-73.

24. Van Norman G, Jackson S, Waisel D. Ethical Issues in Informed Consent. Current Opinions in Anesthesiology 17(2): 177-81, 2004.

25. Van Norman GA. Ethical Issues of Importance to Anesthesiologists Regarding Organ Donation After Cardiac Death. Current Opinion Organ Transplant 10(2): 105-109, 2005.

26. Van Norman GA. Controversies in Organ Donation: Donation After Cardiac Death. Periop Nurs Clin, 3(3):233-240, 2008

27. Van Norman GA. Ethical Issues in Informed Consent. Periop Nurs Clin 3(3):213-222, 2008

28. Ivashkov Y, Van Norman GA. Informed Consent and Ethical Management of the Elderly Patient. Anesthesiology Clinics 2009; 27(3):569-80

29. Souter M, Van Norman GA. Ethical Controversies at End-of-Life After Traumatic Brain Injury: Defining Death and Organ Donation. Crit Care Med 2010; 38(9 suppl): S502-9

30. Souter M, Van Norman GA. [Letter to the Editor] Reply to: Concerns regarding definition of brain death. Critical care medicine 2011;39(3):606-7

31. Sara Kim, PhD; Sinan Jabori, BS; Jessica O'Connell, MD, FACS; Shanna Freeman, APRN; Cha Chi Fung, PhD; Sahrish Ekram, BA; Amruta Unawame, MD; Gail Van Norman, MD. Current Trends of Research Methodologies in Informed Consent Studies: Time to Re-examine? Patient Educ Couns 2013; 93:559-66

32. Van Norman G. Physician Aid-in-Dying: Cautionary Words. Curr Opinion Anesthesiol 2014; 27:177-82

33. Van Norman GA. Five Days at Memorial: Life and Death in a Storm-Ravaged Hospital. Book Review. Anesth Analg 2014; 199:494

34. Van Norman GA. Abusive and Disruptive Behavior in the Surgical Team. *AMA Journal of Ethics*. 2015; 17:215-220. . http://journalofethics.ama-assn.org/2015/03/ecas3-1503.html. Accessed March 3, 2015.

35. Van Norman GA. A Matter of Mice and Men: Ethical Controversies in Animal Experimentation. Int Anesthesiol Clin. 2015; 53:63-78.

36. Rooke GA, Lombaard SA, Dziersk J, Natrajan KM, Van Norman G, Larson LW, Poole JE. Initial experience of an anesthesiology-based service for perioperative management of pacemakers and implantable cardioverter defibrillators. Anesthesiology 2015; 123:1024-32.

37. Nair BG, Grunzweig K, Peterson GN, Horibe M, Nerdilek MB, Newman SF, Van Norman G, et al. Intraoperative blood glucose management: impact of a real-time decision support system on adherence to institutional protocol. J Clin Monitor Comput June 12, 2015; epub ahead of print

38. Grunzweig K, Nair BG, Peterson GN, Horibe M, Neirdilek MB, Newman SF, Van Norman G, et al. Decisional practices and patterns of intraoperative glucose management in an academic medical center. 2016; 32:214-23

39. Rooke GA, Lombaard SA, Van Norman GA, Dziersk J, Natrajan KM, Larson L, Poole JE. In Reply [re: . Initial experience of an anesthesiology-based service for perioperative management of pacemakers and implantable cardioverter defibrillators]. Anesthesiology 2016; 124:1195

40. Van Norman G. Drugs, devices and the FDA: an overview of the approval processes: Part 1 Approval of drugs. JACC Basic Translation Sci 2016; 1: 170-9

41. Van Norman G. Drugs, devices and the FDA: an overview of the approval processes: Part 2 Approval of medical devices. JACC Basic Translation Sci 2016; 1:277-87

42. Van Norman G. Drugs and Devices Part 3: a Comparison of U.S. and European Processes. JACC Basic Translation Sci 2016; 1:399-412

43. Van Norman GA. Decisions regarding foregoing life-sustaining treatments. Curr Opin Anesth 2016; Nov 30 [epub ahead of print].

44. Van Norman GA, Eisenkott R. Technology Transfer: From the Research Bench to Commercialization. Part 1. Intellectual Property Rights-Basics of Patents and Copyrights. JACC Basic Translation Sci 2017; 2:85-97

45. Van Norman GA, Eisenkott R. Technology Transfer. Part 2. The Commercialization Process. JACC Basic Translation Sci 2017; 2:197-208

46. Van Norman GA. Overcoming the declining trends in innovation and investment in cardiovascular therapeutics: beyond EROOM's law. JACC Basic Translational Sci 2017; 2:613-25

47. Van Norman GA. Expanding Patient Access to Investigational Drugs: Single Patient INDs and "Right to Try". JACC Basic Translational Sci 2018; 3:280-91

48. Van Norman GA. Expanding Patient Access to Investigational Drugs: Overview of Intermediate and Widespread Treatment INDs, and Emergency Authorization in Public Health Emergencies. JACC Basic Translational Sci 2018; 3:403-14

49. Van Norman GA. Expanded patient access to investigational new devices: review of emergency and non-emergency expanded use, custom and 3D printed devices. JACC Basic Translational Sci. 2018; 3:533-44

50. Shah AC, Ma K, Faraoni D, Oh D, Rooke A, Van Norman GA. Self-reported functional status predicts post-operative outcomes I noncardiac surgery patients with pulmonary hypertension. PLOS ONE. Aug 16, 2018. https://doi.org/10.1371/journal.pone.0201914

51. Van Norman GA. The problem of Phase II clinical trials: reducing costs and predicting success. JACC Basic Translational Sci 2019; 4:428-37

52. Van Norman GA. Limitations of animal studies for predicting toxicity in clinical trials: part 1: is it time to rethink our current approach? JACC Basic Transl Sci. 2019; 4:845-54

53. Suhr W, Van Norman GA. Anesthesia at the Edge of Life. Ethical issues in organ transplantation at end of life: defining death. Anesthesiol Clin 2020; 38:231-46

54. Van Norman GA, Jackson S. The anesthesiologist and global climate change: an ethical obligation to act. Curr Opin Anesth 2020; 33:577-83.

55. Van Norman GA. Limitations of animal studies for predicting toxicity in clinical trials part 2: potential alternatives to the use of animals in preclinical trials. JACC Basic Transl Sci 2020; 5:387-97

56. Van Norman GA. "Warp Speed" operations in the COVID-19 pandemic: moving too quickly? JACC Basic Trans Sci 2020; 5:730-4

57. Van Norman GA. Update to Drugs, Devices and the FDA. How recent legislative changes have impacted approval of new therapies. JACC Basic Transl Sci. 2020; 5:831-9

58. Van Norman GA. Translational perspective. Decentralized clinical trials: the future of medical product development? J Am Coll Cardiol Basic Trans Science 2021; 6:384-7s

59. Van Norman GA. False "beneficence": physician involvement in executions and torture. Encyclopedia of Life Sciences. Clarke A. Ed. John Wiley & Sons, Inc. In Press, anticipated publication late 2021.

60. Van Norman GA. Data safety and monitoring boards should be required for both early and late phase clinical trials. JACC Basic Trans Sci 2021; 6:887-96

61. Van Norman GA. Ethical Aspects of Declaring Brain Death in Adults. Elsevier Clinical Overviews. (Web-based point-of-care publication, peer-reviewed) Accepted November 2021, anticipate publication early 2022
62. Van Norman GA. Brain Death Declaration in Pediatric Patients. Elsevier Clinical Overviews. Accepted January 2022, anticipate publication S

Books:

Cambridge Textbook of Clinical Ethics for Anesthesiologists. Van Norman G, Ed. Palmer S, Jackson S, Rosenbaum S co-ed. Cambridge University Press, 2011. London, UK.

Book Chapters:

1. Van Norman, G. Jehovah's Witnesses, in Atlee, J. (ed): *Complications in Anesthesiology.* WB Saunders, Philadelphia., 1999, pp. 937-939.

2. Van Norman, G. Patient Confidentiality, in Atlee, J. (ed): *Complications in Anesthesiology.* WB Saunders, Philadelphia, 1999. Pp. 931-933.

3. Van Norman, G. DNR in the Operating Room, in Atlee, J (ed): *Complications in Anesthesiology.* WB Saunders, Philadelphia, 1999, pp. 934-936.

4. Van Norman G. Ethical Considerations: Informed Consent, Advanced Directives, DNR Orders. In Hanson, E.(ed): *Medical Clerkship Companion 2.* Harcourt Brace, Chestnut Hill, MA, 2003.

5. Van Norman G. Ethical Decisions/End-of-Life Care in Patients with Vascular Disease. In Kaplan, J. (ed): *Vascular Anesthesia, 2nd Edition.* Churchill Livingstone, Philadelphia, PA, 2004, pp. 387-406.

6. Van Norman, G. DNR in the Operating Room. In Atlee, J. (ed): *Complications in Anesthesiology, 2nd Edition.* WB Saunders, Philadelphia. 2005.

7. Van Norman, G. Jehovah's Witnesses. In Atlee, J. (ed): *Complications in Anesthesiology, 2nd Edition.* WB Saunders, Philadelphia, 2005.

8. Van Norman, G. Patient Confidentiality. In Atlee, J. (ed): *Complications in Anesthesiology, 2nd Edition.* WB Saunders, Philadelphia, 2005.

9. Van Norman GA. Anesthesiology Ethics. *IN* Singer P, Viens A (eds): *The Cambridge Textbook of Clinical Ethics.* 2008. Cambridge University Press, London.

10. Van Norman GA, Rosenbaum S. Ethical Issues in Anesthesia Care. *IN* Miller R, Ed. *Miller's Anesthesia, 7th Ed.* Elsevier Publications, Philadelphia PA. 2009

11. Van Norman GA. Informed Consent: Respecting Patient Autonomy. *IN* Van Norman GA, Ed. *Cambridge Textbook of Ethics for Anesthesiologists.* 2011 Cambridge University Press, London, UK.

12. Van Norman, GA. Informed Consent for Preoperative Testing: Pregnancy Testing and Other Tests Involving Sensitive Patient Issues. *IN* Van Norman GA, Ed. 2011 *Cambridge Textbook of Ethics for Anesthesiologists.* Cambridge University Press, London, UK.

13. Van Norman GA. Revising the Anatomical Gift Act—the Role of Physicians in Shaping Legislation. *IN* Van Norman GA, Ed. 2011 *Cambridge Textbook of Ethics for Anesthesiologists.* Cambridge University Press, London, UK.

14. Van Norman GA. Animal Subjects Research Part II: Ethics of Animal Experimentation. *IN* Van Norman GA, Ed. 2011 *Cambridge Textbook of Ethics for Anesthesiologists.* Cambridge University Press, London, UK.

15. Van Norman GA. Publication Ethics: Obligations of Authors, Peer-Reviewers and Editors. *IN* Van Norman GA, Ed. 2011 *Cambridge Textbook of Ethics for Anesthesiologists.* Cambridge University Press, London, UK.

16. Van Norman GA. Sexual Harassment, Discrimination, and Faculty-Student Intimate Relationships in Anesthesia Practice. *IN* Van Norman GA, Ed. 2011 *Cambridge Textbook of Ethics for Anesthesiologists.* Cambridge University Press, London, UK..

17. Van Norman GA. Physician Participation in Executions. *IN* Van Norman GA, Ed. 2011 *Cambridge Textbook of Ethics for Anesthesiologists.* Cambridge University Press, London, UK.

18. Wako E, Van Norman GA. Laboratory Testing in Spine Disease. *IN* Chapman JR, Dettori JR, Norvell, DC (2011) *Measurements in Spine Care.* 1st ed. Stuttgart New York: Thieme.

19. Van Norman GA. Ethical Standards in Medical Practice. *IN* Manchikanti L, Christo P, Trescot A, Falco FJE (eds). *Foundations of Pain Medicine and Interventional Pain Management Board Review.* 2011 ASIPP Publishing, Paducah, KY 42001

20. Van Norman GA. Ethics of Research in Pain Management. *IN* Manchikanti L, Christo P, Trescot A, Falco FJE (eds). 2011 *Foundations of Pain Medicine and Interventional Pain Management Board Review.* ASIPP Publishing, Paducah, KY 4200

21. Van Norman GA. Ethics in Anesthesiology. *IN* Miller R, Ed. *Miller's Anesthesia, 8th Ed.* Elsevier Publications, Philadelphia PA, 2015.

22. Van Norman GA. Anesthesia Pearls. *IN* Wong C, Hamlin N Eds. *The Medicine Consult Handbook.* Springer Science and Business Media Inc. New York, NY. 2012

23. Van Norman GA. Organ Transplantation. *IN* Brennan. Michael (ed.). The A-Z of Death and Dying: Social, Medical and Cultural Aspects. Santa Barbara, CA: ABC-Clio. 2013

24. Van Norman GA. Life Support Systems. *IN*: Brennan. Michael (ed.). The A-Z of Death and Dying: Social, Medical and Cultural Aspects. Santa Barbara, CA: ABC-Clio. 2013

25. Jackson S, Van Norman GA. Anesthesia, Anesthesiologists and Modern Medical Ethics. *IN* The Wondrous Story of Anesthesiology, Eger EI II, SAidman L, Westhorpe RN Eds. Springer, NY. 2014 pp205-218

26. Van Norman G, Rosen J. Michael Jackson: Medical Ethics and What Went Wrong. In; Pediatric Sedation Outside of the Operating Room: a Multispecialty International Collaboration, Second Edition. Mason KP Ed. Springer, NY 2015 pp685-698

27. Van Norman, GA. Ethics and clinical aspects of palliative sedation in the terminally ill child. In; Pediatric Sedation Outside of the Operating Room: a Multispecialty International Collaboration, Second Edition. Mason KP Ed. Springer, NY 2015 pp699-710

28. Van Norman G. Anesthesia Pearls. *In* The Perioperative Medicine Consult Handbook, Jackson M, ed. Springer, 2015.

29. Van Norman GA. Preoperative Testing: Ethical Challenges, Evidence-Based Medicine and Informed Consent. In: Ethical Issues in Anesthesiology and Surgery. Springer publications. Jericho B, Ed. Springer, New York, 2016.

30. Van Norman GA. Ethics and Evidence Regarding Animal Subjects Research: Splitting Hares--or Swallowing Camels? In: Ethical Issues in Anesthesiology and Surgery. Jericho B, ed. Springer, New York, 2016.

31. Jackson S, Van Norman GA. Ethics in Research and Publication. In: Ethical Issues in Anesthesiology and Surgery. Jericho B, Ed. Springer, New York. 2016.

32. Van Norman GA. Ethical Issues in Elderly Patients: Informed Consent. In: Barnett S, ed. Perioperative Care of the Elderly Patient. Cambridge University Press, Cambridge UK. 2017

33. Van Norman GA. Ethical Issues: Withdrawing, Withholding and Futility. In Principles of Geriatric Critical Care. Akhtar S, Rosenbaum S, Eds. Cambridge University Press, UK. Dec 6, 2018.

34. Van Norman GA, Rosenbaum S. Ethics in Anesthesiology. Miller's Anesthesia 9th Ed. Elsevier, Philadelphia PA. 2020

35. Van Norman GA, Rosen J. Michael Jackson: Medical Ethics and What Went Wrong. In; Pediatric Sedation Outside of the Operating Room: a Multispecialty International Collaboration, Third Edition. Mason KP Ed. Springer, NY. 2021. pp 813-825

36. Van Norman, GA. Ethical and clinical aspects of palliative sedation of the terminally ill child. In; Pediatric Sedation Outside of the Operating Room: a Multispecialty International Collaboration, Third Edition. Mason KP Ed. Springer, NY. 2021. pp 847-862

37. Van Norman GA. Conscientious Objection in Medical Practice. In: Professional, Ethical, Legal and Educational Lessons in Problem-Based Learning Approach. Oxford University Press, Oxford UK. Accepted. Anticipated publication early 2022.

Other Publications:

Print Publications

1. Van Norman, G. Cheney F. Falsely Elevated Oximeter Reading Dangerous on One Lung. APSF Newsletter (letter to the editor) Issue 23. June, 1989.

2. Van Norman G. Ethics of Informed Consent. ASA Newsletter 58(8): 15-17, 1994

3. Van Norman, G. Special Paper: Implementation of an Ethics Curriculum: Getting Started. In Waisel D, Van Norman G (eds): *ASA Syllabus on Ethics: Informed Consent.* ASA publications, Park Ridge, Illinois. 1997, pp. 1-6.

4. Jackson S, Fine P, Palmer S, Rosebaum S, Truog R, Van Norman G. Letter to Editor, Comment on Bastron, D. Response to: Ethical Concerns in Anesthetic Care for Patients with Do-Not-Resuscitate Orders. Anesthesiology 87(1): 176-177, 1997.

5. Van Norman, G. Who Speaks for the Patient? Ethical Principles in Assessing Patient Competence and Appropriate Use of Proxy Decision-Makers in the Practice of Anesthesiology. In Waisel D, Van Norman G (eds*): ASA Syllabus on Ethics: Informed Consent.* ASA publications, Park Ridge, Illinois. 1997, pp. B2-B34.

6. Waisel D, Van Norman G, Fine P. Special Article, Hospice Care: Live All the Days of Your Life. An Interview with Perry G. Fine. In Waisel D, Van Norman G (eds). *ASA Syllabus on Ethics: Informed Consent.* ASA publications, Park Ridge, Illinois. 1997, pp. 1-9.

7. Van Norman G. Redefining Death. Ethical, Legal and Medical Implications of Brain Death Determination in Anesthesia Practice. In Waisel D., Van Norman G. (eds): *ASA Syllabus on Ethics: End of Life Issues.* ASA publications, Park ridge, Illinois. 1999, pp. G1-G7.

8. Van Norman G. Chapter 17: Misinformed Consent--A Problem in the OR? In*: ASA Refresher Courses in Anesthesiology.* ASA Publications, Chicago Ill. 1999, pp.215-223.

9. Van Norman G, and Posner K. Coronary Stenting or Percutaneous Transluminal Coronary Angioplasty Prior to Noncardiac Surgery Increases Adverse Perioperative Cardiac Events; the Evidence is Mounting. (letter to the editor). J Amer Coll Cardiol 36(7): 2351, 2000.

10. Van Norman, G, Palmer S. When Should Anesthesiologists Restrain Uncooperative Patients? ASA Newsletter 65(3), 2001.

11. Van Norman G. The Student-Teacher Relationship in Medicine: Are Intimate Relationships Between Faculty and Medical Trainees Ethical? In Van Norman G, Waisel D. (eds): *ASA Syllabus on Ethics: Ethics of Professional and Personal Relationships in Anesthesiology Training and Practice.* ASA, Park Ridge, Illinois. 2004.

12. Van Norman G. Non-Heart-Beating Cadaver Organ Donation: Ethical Issues for Anesthesiologists. ASA Newsletter 67(11), 2003.

13. Van Norman GA, Palmer SK, Jackson SH. The Ethical Role of Medical Journal Editors. [Letter] Anesth Analg 100:603-4, 2005.

14. Brown S, Van Norman G. Compassion and Choice in End-of-Life Decisions. Editorials and Opinions, *The Seattle Times*, April 1, 2005

15. Van Norman GA. Practical Ethical Concerns Regarding Intimate Relationships in the Operating Room. ASA Newsletter 2007, 71(5).

16. Van Norman G, Brown S. Organ Donation a Personal Decision. Opinion, *The Seattle Post Intelligencer,* March 20, 2007.

17. Van Norman GA. Contributing Writer: *Handbook of Anesthesia and Co-Existing Disease.* Elsevier publications, Philadelphia PA. 2009.

18. Sweitzer BJ, Vidoga M, Milokjic, et al. Resident's knowledge of ACC/AHA Guidelines for Preoperative Cardiac Evaluation is Limited. Cleveland Clinic J Med 2010; 77(ESuppl):eS11-eS12.

19. Palmer SK, Van Norman GA, Jackson SL. Letter to the editor. Routine pregnancy testing before elective anesthesia is not an American Society of Anesthesiologists standard. Anesth Analg. 2009; 208(5):1715-6.

20. Van Norman GA, Jackson SL. Back to our roots: the importance of enforcing professionalism at the ASA. ASA Newsletter. May, 2011. 75(5):10-12

21. Jackson SL, Van Norman GA. Ethical issues in the publication of medical research. ASA Newsletter. May 2011. 75(5):14-15

22. Van Norman GA. Misinformed Consent: A problem in the Operating Room? Ethical principles of informed consent and their application for the anesthesiologist. Refresher Courses Anesth 2011; 39(1):215-223

23. Van Norman GA. Ethics of Ending Life; Physician-Assisted Suicide and Euthanasia, Part 1. California Society of Anesthesiologists Bulletin, CA. Winter 2012.

24. Van Norman GA. Physician Assisted Suicide. ASA Newsletter. Spring 2012.

25. Van Norman GA. Ethics of Ending Life; Physician-Assisted Suicide and Euthanasia, Part 2. California Society of Anesthesiologists Bulletin, CA. Summer 2012.

26. Van Norman GA. Ethical Challenges of Routine Preoperative Tests. ASA Newsletter, Nov. 2012

27. Rooke A, Natrajan K, Lombaard S, Dziersk J, Van Norman GA, Poole J. Letter to the Editor In Reply: Initial experience of an anesthesiology based service for perioperative management of pacemakers and implantable cardioverter defibrillators. Anesthesiology 2016; 124: 1195

Web publications

1. Update Author, *Sleisinger and Fordtran's Gastrointestinal and Liver Disease, 7th Edition,* on-line version. Mark Feldman MD, Editor. Elsevier Scientific Publications, Philadelphia *[www.sfgastro.com],* 2003 to 2006.

2. Update Author, *Anesthesia, 6th Edition,* on-line version. Ron Miller MD, Editor. Elsevier Scientific Publications, Philadelphia. *[www.anesthesiatext.com]* 2004 to present.

3. Update Author, *Diseases of the Heart, 6th Edition,* on-line version. Eugene Braunwald MD, Editor. Elsevier Scientific Publications, Philadelphia [*www.branwalds.com],* 2004 to present.

4. Update Author, *Murry and Nadel's Textbook of Respiratory Medicine,* on-line version. Robert J. Mason MD, John F. Murray MD, V. Courtney Broaddus MD, and Jay A Nadel MD, editors. Elsevier Scientific Publications, Philadelphia *[www.respmedtext.com],* 2005-2006.

5. Update Author, *Drugs for the Heart,* on-line version. Lionel H Opie MD and Bernard J Gersh MD, editors. Elsevier Scientific Publications, Philadelphia *[www.opiedrugs.com],*, 2005 to 2008

6. Update Author, *Clinical Gastroenterology and Hepatology,* on-line version, Wilfred M Weinstein MD, C J Hawkey MD, J Bosch MD, editors. Elsevier Scientific Publications, Philadelphia *[www.clingastrotext.com],* 2005-2006.

7. Medifile author for *FirstConsult,* Medical website for primary care physicians. Elsevier Scientific Publications, London UK *[www.firstconsult.com],* 1998 to 2008.

8. Topic Editor, *First Consult.* Medical website for primary care physicians. Elsevier Scientific Publications, London UK, *[www.firstconsult.com],* 1998 to 2008

9. Medical writer, *Clinical Procedures*, Elsevier Scientific Publications, PhiladelphiaPA, 2008 to present

10. Medical editor and writer, *Clinical Reviews*, Elsevier Scientific Publications, Philadelphia, 2021

Abstracts:

1. Van Norman G, Pavlin E, Eddy C, and Pavlin J. Hemodynamic and Metabolic Effects of Aortic Unclamping Following Emergency Surgery for Traumatic Thoracic Aortic Tear in Shunted and Unshunted Patients. Presented to the 50th Annual Meeting of the American Association for the Surgery for Trauma, 1989.

2. Van Norman G, Pavlin E, Eddy C, and Pavlin J. Hemodynamic and Metabolic Effects of Aortic Unclamping Following Emergency Surgery for Traumatic Thoracic Aortic Tear in Shunted and Unshunted Patients. Presented to the American Society of Anesthesiologists Annual Meeting, 1989.

3. Van Norman G, Spiess B, Lu J, et al. Aprotinin Versus Aminocaproic Acid in Moderate-to-High-Risk Cardiac Surgery: Relative Efficacy and Costs of Transfusion. Anes Anal Supplement, March 1995.

4. Kenney M, Van Norman G, Hague G, et al. Variations in Ionized Magnesium During Cardiopulmonary Bypass. Presented to the Cardiopulmonary Bypass Meeting, San Diego, California, 1995.

5. Pavlin J, Gunn H, Van Norman G, et al. Optimal Propfol/Alfentanil Combinations for Supplementing N20 For Outpatient Surgery. Presented to the Annual Meeting of the American Society of Anesthesiologists, 1997. Anesthesiology 87(3S) Supplement 308A, 1997.

6. Van Norman G, Posner K, Wright I, et al. Adverse Cardiac Events Following Noncardiac Surgery in Patients with Prior PTCA versus Normal Patients, and Patients with Nonrevascularized CAD. Presented to the Scientific Sessions of the American Heart Association, Orlando, Florida, 1997, published in supplement to Circulation, October 1997.

7. Simmons E, Van Norman G, Robledo J, et al. Effect of Hemofiltration on Aprotinin Activity in Patients Undergoing Cardiopulmonary Bypass. Presented to WARC (Western Anesthesia Residents Conference, 1998.

8. Lee J, Karjeker S, Van Norman GA et al. Advance Directives in the Perioperative Period. Presented to WARC (Western Anesthesia Residents Conference), 2009

9. Karjeker S, Van Norman G, et al. Advance Directives in the PreAnesthesia Clinic. Presented at the American Society of Anesthesiologists' Annual Meeting, New Orleans, LA. 2009

10. Rooke, G.A., Natrajan, K., Lombaard, S., Dziersk, J., Van Norman, G., Poole, J.: Initial experience of an anesthesia-based service for perioperative management of CIEDs. Anesthesiology 117:A835, 2012.
11. Shah A, Ma K, Rooke GA, Van Norman G. Pulmonary HTN in the perioperative patient. (working title). Accepted to IARS Annual Meeting, Honolulu HI. March 2015.

12. Rooke A, Natrajan K, Lombaard S, Dziersk J, Van Norman G, Poole J. Poster: Initial experience of an anesthesia-based service for perioperative management of CIEDs. ASA American Society of Anesthesiologists Annual Meeting, Washington DC, 2012

OTHER

International and National Invitational Lectures:

1. "DNR in the OR: Am I a Bad Doctor if I Let My Patient Die?" American Society of Anesthesiologists, Refresher Course, San Francisco, CA, June 29, 1996.

2. "Misinformed Consent: Is This a Problem in the Operating Room?" American Society of Anesthesiologists Refresher Course, San Francisco, CA, June 29, 1996.

3. "Ethics: A New Hot Topic in Resident Education," The Society for Education in Anesthesia Fall Meeting: Educational Strategies for the 21st Century, New Orleans, 1996.

4. "Ethics: A New Hot Topic in Resident Education," The Society for Education in Anesthesia Fall Meeting: Educational Strategies for the 21st Century, San Diego, CA, October 1997.

5. "Misinformed Consent: A Problem in the Operating Room?" American Society of Anesthesiologists Workshop in Practical Bioethics for the Anesthesiologist, Boston, MA, 1997.

6. "Brain Dead, or Only Mostly Dead? What's the Difference, I'm Just the Anesthesiologist!" American Society of Anesthesiologists Workshop in Practical Bioethics for the Anesthesiologist, Boston, MA, 1997.

7. "PTCA prior to Noncardiac Surgery." World Congress of Cardiovascular Anesthesiologists, Santiago, Chile, 1998.

8. "Ethics Case Discussion: Assessing Cardiac Risks for Patients with Coronary Artery Disease Undergoing Noncardiac Surgery" and "Ethics Case Discussion: Assessing Brain Death" Rush-Presbyterian-St. Luke's Medical Center, Chicago Illinois, Visiting Professorship, 1998

9. "Brain Death: What Every Anesthesiologist Should Know" Midwest Anesthesia Conference and Peri-Anesthesia Care Symposium, Chicago, Il, 1999.

10. "Ethical Issues in the Operating Room," American Society of Anesthesia Technologists and Technicians, Seattle, WA, 2000.

11. "Ethical Issues in the Operating Room," American Society of Extracorporeal Technologists, Seattle, WA, 2000.

12. "Ethical Boundaries of Persuasion: Coercion and Restraint in Pediatric Anesthesia Practice," Mid-Year SAMBA meeting, New Orleans, LA, 2001.

13. "Ethics: Brain Death and Organ Donation" Rush-Presbyterian-St. Luke's Medical Center and Rush University, Chicago, Illinois Department of Anesthesiology and Undergraduate Medical School; Inaugural Speaker, Katalin Selemczi MD Memorial Lecture Series in 2001

14. "Donation After Cardiac Death—Stretching the Definitions of Death Too Far?" Invited lecturer; European Society of Anesthesiology, Copenhagen Denmark, May 2008.

15. "Conflicts of Interest: Industry Reps." February 2009; Visiting Professor, University of Oklahoma Department of Anesthesiology. Oklahoma City, OK

16. "DNR in the Patient Undergoing Surgery and Anesthesia." Sept 2009 Primary Care Medicine. Seattle, WA

17. Perioperative Beta Blockers." National Association of Nurse Practitioners. Aug 2010. Seattle, WA

18. Ghosts of OR Cancellations Past and Present." Combined WSSA, BCAA international meeting. Dec 2010 (Elizabeth Wako MD and Gail Van Norman MD speakers)

19. Ethics of Organ Donation After Cardiac Death. Society for Cardiovascular Anesthesiologist. Annual Meeting, April 2011. Savannah, Georgia.

20. Ethics of Organ Donation After Cardiac Death. Dept of Anaesthesiology and Intensive Care, St. Mary's Hospital. London, UK. August 2011.

21. Physician-assisted Suicide and Euthanasia. Brocher Foundation. Hermance, Switzerland, August 2011.

22. Fraud in Publication and Medical Research. Dept of Anaesthesiology and Intensive Care Medicine, University of Geneva. Geneva, Switzerland. Sept 2011.

23. Informed Consent and Informed Refusal in the Operating Room. AORN annual meeting, Seattle, WA Jan. 2012

24. Fraud and Plagiarism in Research. Risk Management Division, MD Anderson Medical Center, Houston TX, April 18, 2012

25. Physician-Assisted Suicide and Euthanasia, Department of Anesthesiology, MD Anderson Medical Center, Houston TX, April 18, 2012

26. Ethics in Anesthesiology. Annual Review Course for Certified Nurse Anesthetists. Orlando Florida, November 2012

27. Invited Lecturer: Ethics of Interventional Pain Management. ASIPP. Phoenix, AZ. Feb 2012

28. Invited Lecturer: Ethics of Interventional Pain Management. ASIPP. San Francisco CA Aug 2012.

29. Invited Lecturer: Ethics of Interventional Pain Management. ASIPP. Phoenix, AZ. July 2013

30. Invited Lecturer: Ethics of Interventional Pain Management. ASIPP. Denver CO; Oct 2013

31. Invited Lecturer; Controversial Cases in Organ Transplantation: clinical forum. American Society of Anesthesiologists Annual Meeting. San Francisco CA. Oct 2013

32. Invited Lecture: Ethics of DCD Organ Donation. St. Mary's Hospital Dept of Anesthesiology and Critical Care, London UK. Dec 2014

33. Invited Lecture: DNR in the OR. London Soc Anesthesiology, UK. Dec 2014

34. International Academy of Law and Mental Health. Moderator: Brain Death, Personhood, Body Integrity: Ethical and Legal Considerations in Vital Organ Transplantation. Vienna Austria July 2015

35. Invited Lecturer: Harvard Anesthesia Update Spring 2015. Pro/Con Debate: Should Physicians Participate in Lethal Injection.

36. Invited Lecturer: Ethics of Interventional Pain Management. ASIPP, Chicao Il. July, 2015

37. Keynote Speaker: Terminal Sedation in Pediatric Sedation: Suffering, Palliation and Transcendance (working title). Conference for Pediatric Sedation Outside of the Operating Room. Cancun, Mexico Sept 2015

38. Invited Ethics Lecturer: Moya Review Course for Nurse Anesthetists. Orlando Fl, November 2015

39. Invited Participant; 2015 Alumni Meeting of the Scholars of the Brocher Foundation, Geneva Switzerland, June 16-18, 2015.

40. Invited Speaker: 2016 World Congress of Anesthesiologists. Ethics Section. Hong Kong, Aug 27-Sept 1, 2016

41. Invited Lecturer; Medical Professionalism. St. Mary's Hospital Department of Anesthesiology and Critical Care. London, UK. July 2016

42. 2017 International Academy of Law and Mental Health. Invited panelist, moderator. Ethics of Psychosurgery. Prague, Czech Republic, July 2017

43. 2017 Mazama Spine Summit, Mazama WA. Invited Speaker: What is Professionalism and the Practice of Medicine? Lessons from the Michael Jackson Case.

44. 2019 International Academy of Law and Mental Health. Invited panelist, moderator. Physician Assisted Suicide in the United States: Current Status. Rome, Italy. July 2019.

45. Brocher Foundation Reunion of Scholars, 2019. Lethal Injection in the United States: personal experience as an expert witness for the prisoner. Geneva, Switzerland. June 2019

46. 2022 International Academy of Law and Mental Health. Invited moderator and lecturer. Brain Death: does the concept still work? Scheduled for July 21, 2022. Lyon, France.

Regional Invitational Lectures:

1. "Treatment of Intraoperative Emergencies: Wheezing; Inability to Ventilate" CME Lecture, Washington State Society of Anesthesiologists, Seattle, WA, 1991.

2. "Ethical Issues in Anesthesiology," Washington State Society of Anesthesiologists, Moderator and CME Lecturer, Seattle, WA, 1993.

3. Washington State Association of Nurse Anesthetists, Tukwila, Washington "Ethical Issues in Anesthesia Practice", 1995.

4. Washington State Association of Nurse Anesthetists "Ethical Issues in Anesthesia Practice" Seattle, WA, 1998.

5. "Management Issues In the Preoperative Clinic." Society for Ambulatory Anesthesia (SAMBA), Seattle, Washington, 1999.

6. "Physiology of Perioperative Myocardial Blood Flow." Washington State Society of Nurse Anesthetists, Seattle, WA, 1999.

7. "Ethical Issues in the Operating Room." American Society of Anesthesiology Technologists, Seattle, WA, 2000.

8. "DNR orders in the Operating Room," Combined Anesthesia and Surgery Grand Rounds, Southwest Washington Medical Center, Vancouver, WA, 2001.

9. "Medical Ethics: Balancing Patient Advocacy and Managed Care." Western Pension and Benefits Conference, Seattle, WA, 2003.

10. "Conscious Sedation for Radiological Procedures in the Outpatient," Northwest Hospital Radiology Department, Seattle, WA, 1991.

11. Instructor, Certified Post Anesthesia Nurse (CPAN) Certification Course, Northwest Hospital, Seattle, WA, 1991.

12. Conflicts of Interest with Industry. AORN winter meeting, Kent WA, 2009

13. "Preoperative Testing." Wash. State Nurse Anesthetists. Sept 2009, Spokane, WA

13. "Implementing Intra-Operative Glucose Control: What Does it Take?" Washington State Hospital Association. April 2014, Seattle, WA

14. "Who's Doing Your Surgery and Anesthesia? Ethical Issues in Informed Consent in Medical Direction and Overlapping Surgeries." Washington Ambulatory Surgery Association 2018 Conference. November 2018, Seattle WA.

15. The MAD physician and why he is a constant danger to your patients and your institution. Ethical management of the abusive physician in the operating room. Washington Ambulatory Surgery Association 2019 Conference. November 2019, Everett WA.

16. Would You Like Some Fish with Your Propofol? Reducing environmental contamination from the Operating Room. Washington Ambulatory Surgery Association Conference, November 2021

Invited Journal Reviews:

1. Invited Journal Reviewer, cardiovascular anesthesia, *Anesthesia and Analgesia,* 1998 to present.
2. Invited Journal Reviewer, medical ethics, *Anesthesiology*, 1998 to present.
3. Invited Journal Reviewer, medical ethics, *Journal of Obstetrics and Gynecology*, 1998.
4. Clinical Reviewer, *FirstConsult.* Medical website for primary care physicians, Elsevier publications, London UK. *[www.firstconsult.com]* ,1998 to present.
5. Invited Journal Reviewer, medical ethics, *Mayo Clinic Proceedings,* 2006-2009.

6. Invited Journal Reviewer, *Journal of Philosophy, Ethics, and Humanities*, 2007.
7. Invited Journal Reviewer, *European Journal of Anaesthesiology,* 2013
8. Invited Journal Reviewer, *PLos One,* 2021

Web Authorships

1. Update Author, *Anesthesia, 6th Edition,* on-line version. Ron Miller MD, Editor. Elsevier Scientific Publications, Philadelphia. *[www.anesthesiatext.com]* 2004 to present.

2. Update Author, *Sleisinger and Fordtran's Gastrointestinal and Liver Disease, 7th Edition,* on-line version. Mark Feldman MD, Editor. Elsevier Scientific Publications, Philadelphia *[www.sfgastro.com],* 2003 to 2006.

3. Update Author, *Diseases of the Heart, 6th Edition,* on-line version. Eugene Braunwald MD, Editor. Elsevier Publications, Philadelphia [*www.branwalds.com],* 2004 to present.

4. Update Author, *Murray and Nadel's Textbook of Respiratory Medicine,* on-line version. Robert J. Mason MD, John F. Murray MD, V. Courtney Broaddus MD, and Jay A Nadel MD, editors. Elsevier publications, Philadelphia *[www.respmedtext.com],* 2005-2006.

5. Update Author, *Drugs for the Heart,* on-line version. Lionel H Opie MD and Bernard J Gersh MD, editors. Elsevier publications, Philadelphia *[www.opiedurgs.com],* 2005 to present.

6. Update Author, *Clinical Gastroenterology and Hepatology,* on-line version, Wilfred M Weinstein MD, C J Hawkey MD, J Bosch MD, editors. Elsevier publications, Philadelphia *[www.clingastrotext.com],* 2005-2006

7. Medifile Author for *FirstConsult*, Medical website for primary care physicians. Elsevier publications, London UK *[www.firstconsult.com],* 1998 to 2006

8. Medical Writer, *Procedures Consult*, Anesthesia Procedures. Elsevier publications, Philadelphia, PA. 2007-2008

9. Review and update Procedures Consult for Anesthesia, Surgical, Cardiovascular, and Primary Care Procedures. Elsevier publications, Philadelphia PA. 2020-2021

Miscellaneous:

Consulting Anesthesiologist, Woodland Park Zoological Society, 1992-2002.
Medical writer Handbook for Stoelting's Anesthesia and Co-Existing Disease, 3rd Edition 2009
Content/formatting editor, Journal of American College of Cardiology 2015-2020
Medical Writer, Journal of American College of Cardiology 2016-present

Updated, January 2022

Appendix II: Gail Van Norman MD Depositions and Court Testimony in the Last 4 Years

1. **2018/2019:** Jason McGehee, et al. v. ASA Hutchinson et al. US District Court, Eastern District of Arkansas, Little Rock Division. **Testimony for the plaintiffs (prisoners).** Capital punishment case. Expert testimony regarding lethal injection drugs. Defendants prevailed.

2. **2018. Wickland V. Swedish Health Services, Swedish Neuroscience Institute and Johnny B Delashaw MD** (Seattle, WA). **For plaintiff (patient). Deposition** given regarding informed consent, concurrent surgical cases, and obligation of colleagues and institutions to control abusive behavior in the operating room. Case settled in favor of plaintiff.

3. **2018 : James S Goff MD v. State of Washington, Department of Social and Health Services. For the plaintiff (physician).** Case of a physician (Goff) regarding alleged negligent investigation revocation of license over fraudulent criminal complaints against the physician that were later proven to be false, and to have further been proven in court to have been falsified by a state investigator. **Deposition** given regarding career impact on physicians arising out of the negligent process. Verdict in favor of plaintiff.

4. **2020:** Expert regarding informed consent in human subjects research: **Butler and Holland v. Juno Therapeutics** (Houston, TX). **For plaintiffs (patients): deposition** regarding the standards and federal regulations for informed consent for human subjects in clinical research. Case settled in favor of plaintiffs.

5. **2020: Murdoch v. Swedish Health Services and Johnny B. Deleshaw. (Seattle WA) For plaintiff (patient). Deposition** regarding informed consent, and coercion. Case settled in favor of plaintiff.

6. **2020: EC v. Presbyterian Healthcare Services, et al. (Alberqueque, NM). For plaintiff. (patient). Deposition** regarding postoperative delirium and management of sexual assault in the workplace. Case settled in favor of plaintiff.

7. **2021: Roane, et al. v Barr.** Expert regarding the effects of lethal injection drugs on prisoners undergoing judicial execution. Declaration and testimony before the Washington DC Federal Court of Appeals, January 2021. Case decided in **favor of the plaintiffs (prisoners)**. Overturned by U.S. Supreme Court without hearing.

8. **2021:** Expert testimony for **Kim v. Washington State Medical Commission. For defendant (physician)** Ethics of physician requests and use of patients, employees, models, social partners and others for educational physical examination. Case determination pending.

9. **2022: King Vs. Parker, et al. State of Oklahoma. For plaintiffs. Deposition** regarding lethal injection drugs. Case ongoing.

Appendix III: Materials Relied Upon and/or Reviewed

In addition to materials previously mentioned, I have also reviewed

- Several publications by media eyewitnesses to the execution of inmates involving the use of midazolam as detailed and footnoted in this report.
- Autopsy reports of over 300 prisoners executed in the United States by lethal injection 37 of which were obtained by Noah Caldwell for National Public Radio in 2020 through the Freedom of Information Act. Three additional reports, those of Billy Ray Irick, Donnie Johnson and John Grant, were supplied by their attorneys. This does not represent a complete list of prisoners executed using midazolam, as some of the autopsy files may not have been released, and many prisoners did not have autopsies.
- Published studies, articles, reviews and textbooks and media accounts of executions, including those cited and footnoted in my report and others as noted.
- Materials supplied by attorneys:

    - A document signed by Scott Crow, Director of Oklahoma Department of Corrections, dated 2/20/2020 and beginning "Execution of Inmates Sentenced to Death"
    - Transcript of court testimony of Dr. Joseph Antognini dated. 1/10/2022
    - Transcript of court testimony of Julie Gardner dated 1/10/2022
    - Transcript of court testimony of Meghan LeFranscois dated 1/10/2022
    - Transcript of court testimony of Dr. Michael Weinberger dated 1/10/2022
    - Transcript of court testimony of Dr. Ervin Yen dated 1/10/2022
    - Expert Opinion of Michael Weinberger MD, Case no. CIV-14-665-F dated January 11, 2021
    - Expert report of Dr. Joseph Antognini to the US Western District Court for the Western District of Oklahoma dated January 15, 2021
    - Expert Report of Charles Buffington, PharmD, to the US District Court for the Western District of Oklahoma, No 14-cv-665-F
    - Oklahoma Department of Corrections Service Log Stouffer dated 12/9/21
    - A document disclosed by defendants labeled "Grant OAG - 019353-OAG – 019353": a handwritten document that appears to have been made by a DOC witness containing notes regarding the events in the John Grant execution
    - A file labeled Grant ECG OAC that contains a series of EKG strips from the execution of John Grant.
    - A document entitled Oklahoma Department of Corrections Correctional Service Log OAG - 019037-OAG – 019042 dated 10/28/21:
    - A document entitled ME and Tox reports from the Office of the Chief Medical Examiner containing the autopsy report and toxicology report for John Grant.
    - A file containing photographs entitled "OAG-019643-AG-019742", "OAG-019643-AG-019842", "OAG-019643-AG-019942", OAG-019643-AG-020009" and OAG-020010-OAG-020087" from the John Grant autopsy

- A document entitled: Richard E. Glossip et al. v. Randy Chandler et al. Transcript for motion of preliminary injunction before the Honorable Stephen P. Friot United States District Judge January. 10, 2022
- A document entitled Hahn 2021-10-25 (Doc 537) Pl Hrg Transcript provided by counsel.
- Affadavit of Julie Gardner dated Nov 17, 2021
- Affadavit of Meghan LeFrancois dated Nov 17, 2021

Appendix IV: Prisoners executed using 3-drug midazolam protocols in the United States with autopsy files and measured blood levels of midazolam‡

Name	State	Date of Execution	Site of blood sample/Midazolam drug level (ng/ml)
Banks, Chadwick	FL	11/13/14	Fem /1900
Davis, Eddie	FL	7/10/14	Iliac /1500
Henry, John	FL	6/8/14	Iliac /1700
Hendrix, Robert	FL	3/20/14	Iliac /1000
Henry, Robert	FL	7/10/14	Iliac /1500
Howell, Paul	FL	2/26/14	Iliac /2700
Morva, William Charles	VA	7/6/17	Iliac /2300
Warner, Charles	OK	1/15/15	Femoral /3200
Samra, Michael	AL	5/17/19	Iliac /1600
Ray, Dominique	AL	2/8/19	Cardiac/> 1000
Price, Christopher4er	AL	5/31/19	Iliac/1000
Moody, Walter Leroy	AL	4/19/18	Iliac /1900
Eggers, Michael Wayne	AL	4/19/18	IVC /4000
McNabb, Torey Twayne	AL	10/19/17	Cardiac /4200
Melson, Robert Bryant	AL	6/8/17	No levels reported.
Arthur, Thomas Douglas	AL	5/26/17	No levels reported.
Smith, Ronald Bert	AL	12/8/16	No levels reported.
Brooks, Christopher Eugene	AL	1/21/16	No levels reported.
Williams, Marcel Wayne	AR	5/27/16	Peripheral /950
Williams, Kenneth D	AR	4/28/17	Peripheral /1800
Lee, Lidell	AR	4/21/17	Peripheral /1400
Locket, Clayton Darrell	OK	4/29/13	Femoral /570
Wood, Joseph Rudolf	AZ	7/24/14	Peripheral /3690
Irick, Billy Ray	TN	8/9/18	Femoral /390
Johnson Donnie Edward	TN	5/16/19	Femoral /930
John Grant	OK	10/28/21	Femoral/2200

Mean plasma level of midazolam 1644 ng/ml

Post mortem drug levels pose challenges in interpretation: the levels of a given drug depends on what site the blood is drawn, and whether the drug is one that undergoes post mortem redistribution (PMR) in the blood and body after death. Obviously accurately understanding the levels of blood in a person's body at the time of death is critical in forensic medicine, to determine the cause of death and whether death occurred because of a drug-related misadventure. Some drugs and classes of drugs do not undergo significant PMR, and postmortem levels that are drawn from a standard site in the body reflect antemortem levels. Because blood drawn from the heart may show much higher levels than in the blood or effective site in the body, heart blood and other more central veins are not generally acceptable for forensic purposes, and the standard

sites from which postmortem drug levels is determined are the femoral and iliac veins.[134,135]For this reason, in calculating the mean above, only the cases in which blood was taken from the femoral or iliac veins is included.

Benzodiazepines represent one class of drug that does not appear to have significant PMR. The PM blood/therapeutic plasma concentration relationship is equal to 1 if the postmortem levels are within the average therapeutic range of the drug. Launianen et al. found that for midazolam the ratio is 1.0, for diazepam a closely related drug, the ratio is 0.9.[136] The average time from death to autopsy in their series of approximately 50,000 autopsies was 5 days, which allows significant time for PMR. Smaller intervals are likely to have less effect on PMR. Most autopsies of executed prisoners took place within 24 hours of execution. The blood was drawn from John Grant's body approximately 3 hours after death, for example. Similarly, Mantinieks et al.[137] studied over 800 autopsies in which both ante mortem and post mortem blood levels of drugs were determined and found that benzodiazepines as a class of drugs had very small PMR values compared to other classes of drugs. They concluded that benzodiazepines have low propensity for PMR. Thus the drug levels above likely reflect the drugs levels at time of death.

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[134] Mantinieks D, et al. Postmortem drug redistribution: a compilation of postmorem/antemortem drug concentration ratios. J Analytic Tox 2021; 45:368-77

[135] Launiainen T, Ojanpera I. Drug concentrations in post-mortem femoral blood compared with therapeutic concentrations in plasma. Drug Test Analysis 2014; 6:308-16

[136] Launiainen T, Ojanpera I. Drug concentrations in post-mortem femoral blood compared with therapeutic concentrations in plasma. Drug Test Analysis 2014; 6:308-16

[137] Mantinieks D, et al. Postmortem drug redistribution: a compilation of postmorem/antemortem drug concentration ratios. J Analytic Tox 2021; 45:368-77

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, F-ABFT, DABCC-TC, Laboratory Director

CONFIDENTIAL

Toxicology Report

Report Issued 12/18/2014 11:03

To: **60168**
University of Florida Pathology Laboratories
4800 SW 35th Drive

Gainesville, FL 32608

Patient Name	BANKS, CHADWICK
Patient ID	R14-02237
Chain	11843719
Age 43 Y	**DOB** Not Given
Gender	Male
Workorder	14320232

12-30-14

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	1900	ng/mL	001 - Femoral Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
3057B	Midazolam, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Red Vial	2.75 mL	Not Given	Femoral Blood	

All sample volumes/weights are approximations.

Specimens received on 12/12/2014.

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 07/25/2014 07:01

To: **60168**
University of Florida Pathology Laboratories
4800 SW 35th Drive

Gainesville, FL 32608

Patient Name DAVIS, EDDIE
Patient ID R14-01373
Chain 11767689
Age 45 Y **DOB** Not Given
Gender Male
Workorder 14179996

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	**1500**	**ng/mL**	**001 - Iliac Blood**

See Detailed Findings section for additional information

9.9.2014

Testing Requested:

Analysis Code	Description
3057B	Midazolam, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Red Vial	3 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.

Specimens received on 07/18/2014.

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Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 07/03/2014 15:01

To: **60168**
University of Florida Pathology Laboratories
4800 SW 35th Drive

Gainesville, FL 32608

Patient Name	HENRY, JOHN
Patient ID	R14-01223
Chain	11767674
Age 63 Y	**DOB** Not Given
Gender	Male
Workorder	14161270

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	**1700**	**ng/mL**	**001 - Iliac Blood**

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
3057B	Midazolam, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Red Vial	3 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.

Specimens received on 06/27/2014.

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 05/30/2014 16:01

To: **60168**
University of Florida Pathology Laboratories
4800 SW 35th Drive

Gainesville, FL 32608

Patient Name	HENDRIX, ROBERT
Patient ID	R14-00856
Chain	11727604
Age 47 Y	**DOB** Not Given
Gender	Male
Workorder	14128498

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	**1000**	**ng/mL**	**001 - Iliac Blood**

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
3057B	Midazolam, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Red Vial	3 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.

Specimens received on 05/23/2014.

Received 15 July 2014 MW

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
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e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC-TC, Laboratory Director

CONFIDENTIAL

Toxicology Report

Report Issued 04/03/2014 15:02

To: **60168**
University of Florida Pathology Laboratories
4800 SW 35th Drive

Gainesville, FL 32608

Patient Name	HENRY, ROBERT
Patient ID	R14-00613
Chain	11727575
Age	55 Y **DOB** Not Given
Gender	Male
Workorder	14074355

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	1500	ng/mL	001 - Iliac Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
3057B	Midazolam, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Red Vial	2.8 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.
Specimens received on 03/28/2014.

Reviewed 10 June 2014 — WH

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
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e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 03/06/2014 17:01

To: **60168**
University of Florida Pathology Laboratories
4800 SW 35th Drive

Gainesville, FL 32608

Patient Name	HOWELL, PAUL
Patient ID	R14-00418
Chain	11727572
Age 48 Y	**DOB** Not Given
Gender	Male
Workorder	14050041

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	2700	ng/mL	001 - Iliac Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
3057B	Midazolam, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Red Vial	3 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.
Specimens received on 02/28/2014.

Reviewed 21 April 2014 VH

Commonwealth of Virginia

ORIGINAL

DEPARTMENT OF FORENSIC SCIENCE

CERTIFICATE OF ANALYSIS

Central Laboratory
700 N. 5th Street
Richmond, VA 23219

Tel. No.: (804) 786-4707
Fax: (804) 786-6907

September 15, 2017

TO: LAUREN N. HUDDLE, M.D.
OFFICE OF THE CHIEF MEDICAL EXAMINER
400 EAST JACKSON STREET
RICHMOND, VA 23219

FS Lab #C17-8070

Your Case #: C0455-17, C2017-64386

Victim(s): MORVA, William

Suspect(s): - - -

Evidence Submitted By: Pamela Blizzard Date Received: 07/11/2017

Item TX1 Two (2) red border vials of iliac blood
Item TX2 One (1) vial of vitreous

RESULTS:

Item TX1
Iliac Blood:
Ethanol none detected
Midazolam 2.3 mg/L
alpha-Hydroxymidazolam present

No other drugs and/or drug classes were detected.

Item TX1 was screened for the following drugs and/or drug classes:
Ethanol, methanol, acetone, isopropanol, cocaine/benzoylecgonine, opiates, oxycodone/oxymorphone, methamphetamine/methylenedioxymethamphetamine (MDMA), phencyclidine, barbiturates, benzodiazepines, carisoprodol/meprobamate, fentanyl, methadone, zolpidem, cannabinoids.

Item TX2
Vitreous:
Ethanol none detected

Item TX2 was screened for the following drugs and/or drug classes:
Ethanol, methanol, acetone, isopropanol.

See attached document for Uncertainty of Measurement reporting.

Supporting examination documentation is maintained in the case file. The evidence is being returned to the Office of the Chief Medical Examiner.

BOARD OF MEDICOLEGAL INVESTIGATIONS
OFFICE OF THE CHIEF MEDICAL EXAMINER
901 N.Stonewall
Oklahoma City, Oklahoma 73117

REPORT OF LABORATORY ANALYSIS

OFFICE USE ONLY
Re. _____ Co. _____
I hereby certify that this is a true and correct copy of the original document. Valid only when copy bear im-print by the office seal.
By _____
Date _____

ME CASE NUMBER: 1500255
LABORATORY NUMBER: 150241
DECEDENT'S NAME: CHARLES WARNER
DATE RECEIVED: 1/16/2005
MATERIAL SUBMITTED: BLOOD
HOLD STATUS: 5 YEARS
SUBMITTED BY: KYLA JORGENSON
MEDICAL EXAMINER: JOSHUA LANTER M.D.

NOTES: Duplicate of case 15-0267

ETHYL ALCOHOL:

Blood: NEGATIVE (Femoral)

Vitreous:

Other:

CARBON MONOXIDE

Blood:

TESTS PERFORMED:

ALKALINE DRUG SCREEN - (Femoral Blood)
BENZODIAZEPINES BY LCMS - (Femoral Blood)
EIA - (Femoral Blood) - Amphetamine, Methamphetamine, Fentanyl, Cocaine, Opiates, PCP, Barbiturates, Benzodiazepines
(The EIA panel does not detect Oxycodone, Methadone, Lorazepam or Clonazepam)

RESULTS:

MIDAZOLAM
3.2 mcg/mL - (Femoral Blood)

02/25/2015
DATE

Byron Curtis, Ph.D., F-ABFT, Chief Forensic Toxicologist

NMS LABS

NMS Labs
200 Welsh Road, Horsham, PA 19044-2208
Phone: (215) 657-4900 Fax: (215) 657-2972
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Robert A. Middleberg, PhD, F-ABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 06/09/2019 17:06

To: 20107
Alabama Department of Forensic Science -
Attn: Dr. Curt Harper
2026 Valleydale Road
Hoover, AL 352442095

Patient Name	SAMRA, MICHAEL
Patient ID	19ME01702
Chain	30233382
Age Not Given	**DOB** Not Given
Gender	Not Given
Workorder	19160596

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	1600	ng/mL	001 - Iliac Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
9329B	Benzodiazepines Panel, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Blue Vial	1.75 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.

Specimens received on 05/31/2019.

ALABAMA
DEPARTMENT OF FORENSIC SCIENCES

2026 Valleydale Road
Hoover, AL 35244-2095

Telephone (205) 969-9292
Facsimile (205) 403-2025

TOXICOLOGICAL ANALYSIS REPORT

Staci Turner, MD
Alabama Department of Forensic Sciences
P. O. Box 7925 Crichton Station
2451 Fillingim Street
Mobile, AL 36670-7925

ADFS Case Number 19ME00956
Agency Case Number
Case Date 02/07/2019
Date Completed 05/22/2019
Report ID 120170234

Subject Ray, Dominique

Evidence analyzed (Including sub-items)

Item	Specimen	Analyte	Result	Method(s)	Notes
1S1	Blood, cardiac	Ethanol	Negative	HS/GC	
1S1	Blood, cardiac	Reference laboratory analysis			3
1S1-1S2	Blood, cardiac	Midazolam	Greater than 1000 ng/mL	EIA, GC/MS, LC/MS/MS	
1S3	Blood, cardiac		NA		
1S4	Urine		NA		
1S5	Urine		NA		
1S6	Vitreous humor		NA		

Footnotes

NA - Not analyzed/Not applicable
3 - Analysis was conducted by a reference laboratory; report is attached.

Comments

Evidence was received in a sealed plastic bag.

A complete report includes a 2-page reference report.

Remaining evidence will be disposed 24 months from the date of this report unless storage space becomes limited or alternate arrangements are made prior thereto.

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Toxicology Report

Report Issued 07/01/2019 15:05

To: **20107**
Alabama Department of Forensic Science -
Attn: Dr. Curt Harper
2026 Valleydale Road
Hoover, AL 352442095

Patient Name	PRICE, CHRISTOPHER
Patient ID	19ME01791
Chain	30233392
Age Not Given	**DOB** Not Given
Gender	Not Given
Workorder	19181865

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	1000	ng/mL	001 - Femoral Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
9329B	Benzodiazepines Panel, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Blue Vial	2.75 mL	Not Given	Femoral Blood	

All sample volumes/weights are approximations.

Specimens received on 08/19/2019.

NMS Labs
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e-mail: nms@nmslabs.com
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Toxicology Report

Report Issued 05/30/2018 18:03

To: **20107**
Alabama Department of Forensic Science -
Attn: Dr. Curt Harper
2026 Valleydale Road
Hoover, AL 352442095

Patient Name MOODY, WALTER
Patient ID 18ME01272
Chain 30221479
Age Not Given **DOB** Not Given
Gender Not Given
Workorder 18148905

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	**1900**	**ng/mL**	**001 - Iliac Blood**

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
9329B	Benzodiazepines Panel, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Blue Vial	3.2 mL	Not Given	Iliac Blood	

All sample volumes/weights are approximations.

Specimens received on 05/22/2018.

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, F-ABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 04/25/2018 18:02

To: **20107**
Alabama Department of Forensic Science -
Attn: Dr. Curt Harper
2026 Valleydale Road
Hoover, AL 352442095

Patient Name EGGERS, MICHAEL
Patient ID 18ME01060
Chain 30221463
Age Not Given **DOB** Not Given
Gender Not Given
Workorder 18107030

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	4000	ng/mL	001 - IVC (Inferior Vena Cava) Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
9329B	Benzodiazepines Panel, Blood

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Blue Vial	1.75 mL	Not Given	IVC (Inferior Vena Cava) Blood	

All sample volumes/weights are approximations.

Specimens received on 04/13/2018.

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e-mail:
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Toxicology Report

Report Issued 11/13/2017 15:03

To: **20107**
Alabama Department of Forensic Science -
Attn: Dr. Curt Harper
2026 Valleydale Road
Hoover, AL 352442095

Patient Name	MCNABB, TORREY
Patient ID	18ME00120
Chain	30204696
Age Not Given	**DOB** Not Given
Gender	Not Given
Workorder	17342688

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	4200	ng/mL	001 - Cardiac Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
9329B	Benzodiazepines Panel, Blood

Tests Not Performed:

Part or all of the requested testing was unable to be performed. Refer to the **Analysis Summary and Reporting Limits** section for details.

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Blue Vial	1.8 mL	Not Given	Cardiac Blood	

All sample volumes/weights are approximations.

Specimens received on 11/03/2017.

NMS LABS

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Workorder	17342688
Chain	30204696
Patient ID	18ME00120

Detailed Findings:

Rpt.

RADIOLOGY:

No radiographs were taken.

WITNESSES:

No visitors from outside agencies were present during the examination.

LABORATORY RESULTS

TOXICOLOGY:

Marcel Williams:

Peripheral blood

Volatile

Acetone	not detected	
Ethanol	not detected	
Isopropanol	not detected	
Methanol	not detected	

Immunoassay

Benzodiazepines	positive	
Cannabinoids	negative	
Cocaine	negative	
Methadone	negative	
Methamphetamines	negative	
Opiates	negative	
Oxycodone	negative	
Propoxyphene	negative	

General Toxicology

Caffeine	present	
Midazolam	0.95 µg/mL	(± 0.11 µg/mL)

Note: Reported measurement uncertainties define an interval having a level of confidence of at least 95%.

LABORATORY RESULTS

TOXICOLOGY:

Kenneth Williams:

Peripheral blood

Volatile

Acetone	not detected	
Ethanol	not detected	
Isopropanol	not detected	
Methanol	not detected	

Immunoassay

Benzodiazepines	positive	
Cannabinoids	negative	
Cocaine	negative	
Methadone	negative	
Methamphetamines	negative	
Opiates	negative	
Oxycodone	negative	
Propoxyphene	negative	

General Toxicology

Caffeine	present	
Midazolam	1.8 µg/mL	(± 0.2 µg/mL)

Note: Reported measurement uncertainties define an interval having a level of confidence of at least 95%.

3. The clothing is released to the investigating agency.

SPECIMENS:

1. Blood and urine are submitted for toxicological analysis.
2. Vitreous fluid, peripheral blood, stock tissue, and liver samples are retained for possible future studies.

LABORATORY RESULTS

TOXICOLOGY:

Ledell Lee:

Peripheral blood

Volatile		
Acetone	not detected	
Ethanol	not detected	
Isopropanol	not detected	
Methanol	not detected	
Immunoassay		
Benzodiazepines	positive	
Cannabinoids	negative	
Cocaine	negative	
Methadone	negative	
Methamphetamines	negative	
Opiates	negative	
Oxycodone	negative	
Propoxyphene	negative	
General Toxicology		
Caffeine	present	
Midazolam	1.4 µg/mL	(± 0.2 µg/mL)

The above reported drug amount has not been corroborated by replicate analysis.

Note: Reported measurement uncertainties define an interval having a level of confidence of at least 95%.

BOARD OF MEDICOLEGAL INVESTIGATIONS
OFFICE OF THE CHIEF MEDICAL EXAMINER
901 N.Stonewall
Oklahoma City, Oklahoma 73117

REPORT OF LABORATORY ANALYSIS

OFFICE USE ONLY
Re. ____ Co. ____
I hereby certify that this is a true and correct copy of the original document. Valid only when copy bear im-print by the office seal.
By _____
Date _____

ME CASE NUMBER: 1401959
LABORATORY NUMBER: 141639
DECEDENT'S NAME: CLAYTON D LOCKETT
DATE RECEIVED: 4/30/2014
MATERIAL SUBMITTED: BLOOD
HOLD STATUS: 5 YEARS
SUBMITTED BY: MEGAN HARRIS
MEDICAL EXAMINER: JOSHUA LANTER M.D.

NOTES:

ETHYL ALCOHOL:

Blood: Not Performed

Vitreous:

Other:

CARBON MONOXIDE

Blood:

TESTS PERFORMED:

ALKALINE DRUG SCREEN - (Femoral Blood)
EIA - (Femoral Blood) - Amphetamine, Methamphetamine, Fentanyl, Cocaine, Opiates, PCP, Barbiturates, Benzodiazepines
(The EIA panel does not detect Oxycodone, Methadone, Lorazepam or Clonazepam)

RESULTS:

MIDAZOLAM
0.57 mcg/mL - (Femoral Blood)

Lidocaine
Detected (Not Confirmed) - (Femoral Blood)

MAY 15 2014

05/14/2014
DATE

Byron Curtis, Ph.D., DABFT, Chief Forensic Toxicologist

AIT

2265 Executive Drive, Indianapolis, IN 46241
Telephone: (800)875-3894 / Fax: (317)243-2789
Toxicology: (317)381-5678

Laboratory Specimen No: 40431418 **Continued..**

Analyte Name	Result	Concentration	Units	Therapeutic Range	Loc
Midazolam	POSITIVE				
Midazolam, Quant		3690	ng/mL	50 - 600	
ANTICONVULSANTS	Negative				
ANTIDEPRESSANTS	Negative				
ANTIHISTAMINES	Negative				
ANTIPSYCHOTICS	Negative				
CARDIOVASCULAR AGENTS	Negative				
ENDOCRINE AGENTS	Negative				
GASTROENTEROLOGY AGENTS	Negative				
NARCOTICS	Negative				
NEUROLOGY AGENTS	Negative				
SEDATIVES/HYPNOTICS	Negative				
STIMULANTS	POSITIVE				
Caffeine	POSITIVE				

Specimens will be kept for at least one year from the date of initial report.

GN 8/21/14

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, F-ABFT, DABCC-TC, Laboratory Director

CONFIDENTIAL

Supplemental Report

Report Issued 11/26/2018 12:00
Last Report Issued 09/07/2018 07:02

To: **10341**
Forensic Medical Management Services - Nashville
850 R.S. Gass Blvd.

Nashville, TN 37216

Patient Name	IRICK, BILLY RAY
Patient ID	MEC#18-2174
Chain	18254411
Age Not Given	**DOB** Not Given
Gender	Not Given
Workorder	18254411

Positive Findings:

Compound	Result	Units	Matrix Source
Caffeine	Positive	mcg/mL	001 - Femoral Blood
Midazolam	390	ng/mL	001 - Femoral Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
8042B	Postmortem, Expanded w/Vitreous Alcohol Confirmation, Blood (Forensic)

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Gray Top Tube	9.3 mL	08/10/2018 13:38	Femoral Blood	
002	Red Top Tube	4.1 mL	08/10/2018 13:38	Vitreous Fluid	
003	White Plastic Container	26 mL	08/10/2018 13:38	Urine	

All sample volumes/weights are approximations.

Specimens received on 08/31/2018.

NMS

NMS Labs
200 Welsh Road, Horsham, PA 19044-2208
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, F-ABFT, DABCC-TC, Laboratory Director

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Toxicology Report

Report Issued 06/05/2019 10:04

To: 10341
Forensic Medical Management Services - Nashville
850 R.S. Gass Blvd.

Nashville, TN 37216

Patient Name	JOHNSON, DONNIE
Patient ID	MEC# 19-1293
Chain	19150044
Age Not Given	**DOB** Not Given
Gender	Not Given
Workorder	19150044

Positive Findings:

Compound	Result	Units	Matrix Source
Midazolam	930	ng/mL	001 - Femoral Blood
Glipizide	360	ng/mL	001 - Femoral Blood

See Detailed Findings section for additional information

Testing Requested:

Analysis Code	Description
8042B	Postmortem, Expanded w/Vitreous Alcohol Confirmation, Blood (Forensic)

Specimens Received:

ID	Tube/Container	Volume/ Mass	Collection Date/Time	Matrix Source	Miscellaneous Information
001	Gray Top Tube	10 mL	05/17/2019 09:00	Femoral Blood	
002	Gray Top Tube	9.5 mL	05/17/2019 09:00	Heart Blood	
003	Red Top Tube	7.25 mL	05/17/2019 09:00	Vitreous Fluid	
004	White Plastic Container	60 mL	05/17/2019 09:05	Urine	

All sample volumes/weights are approximations.

Specimens received on 05/21/2019.

BOARD OF MEDICOLEGAL INVESTIGATIONS
OFFICE OF THE CHIEF MEDICAL EXAMINER
921 N.E. 23rd St
Oklahoma City, OK 73105

REPORT OF LABORATORY ANALYSIS

OFFICE USE ONLY
Re. ____ Co. ____
I hereby certify that this is a true and correct copy of the original document. Valid only when copy bear im-print by the office seal.
By _____
Date _____

ME CASE NUMBER: 2107045
LABORATORY NUMBER: 215451
DECEDENT'S NAME: JOHN MARION GRANT
DATE RECEIVED: 11/1/2021
MATERIAL SUBMITTED: BLOOD, VITREOUS, URINE, LIVER, BRAIN, GASTRIC, BILE, MUSCLE
HOLD STATUS: 5 YEARS
SUBMITTED BY: SARAH CAMPBELL
MEDICAL EXAMINER: JEREMY SHELTON M.D.

NOTES:

ETHYL ALCOHOL:

Blood: Not Performed

Vitreous:

Other:

CARBON MONOXIDE

Blood:

TESTS PERFORMED:

ALKALINE DRUG SCREEN - (Heart Blood)
BENZODIAZEPINES BY LCMS - (Heart Blood)
EIA - (Femoral Blood) - Amphetamine, Methamphetamine, Fentanyl, Cocaine, Opiates, PCP, Barbiturates, Benzodiazepines
(The EIA panel does not detect Oxycodone, Methadone, or Clonazepam)

RESULTS:

ALPRAZOLAM
POSITIVE - (Less than 25 ng/mL) - (Femoral Blood)

MIDAZOLAM
2200 ng/mL - (Femoral Blood)

Jesse M. Kemp PhD

01/06/2022
DATE

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